

Motor nervous system

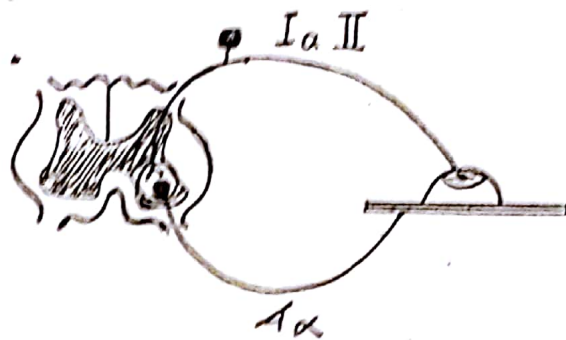
Spinal reflexes i.e. centre is sp. cd.

- 1 Mono synaptic i.e. no interneurone Stretch reflex.
- 2 Polysynaptic, i.e. one or more , Other reflexes.

Stretch (myotatic) reflex

Moderate stretch Reflex → contraction.

- Stimulus: Moderate stretch.
- Receptor: Muscle spindle.
- Afferent: Ia & II
- Centre: Sp. cd.
- Efferent: $A\alpha$
- Response: Contraction.



Object To maintain muscle LENGTH constant (feedback).

Structure of muscle spindle

- Each m. spindle contains 8-10 intrafusal m.f. (smaller & parallel).
- Two types :
 - 1 Nuclear bag Dynamic receptor Rapidly adapting.
 - 2 Nuclear chain Static receptor Slowly adapting.

i.e. discharge at increased rate so long as the muscle is stretched

Central part : Receptor Peripheral part Contractile
- Two afferents : From central part
 - 1 Primary (annulospiral) Ia (16μ 120 m/sec) from Bag & Chain.
 - 2 Secondary (flower spray) II (8μ) from Chain.
- Two efferents : Supply peripheral part & neurones 4μ
 - 1 Dynamic & (Plate endings) to nuclear BAG . CNS
 - 2 Static & (Trail endings) to nuclear CHAIN . ①

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- NB 1 Extrafusal ordinary contractile m.fs are supplied by A alpha
- 2 Muscle spindle capsule is attached to tendon or sides of extrafusal
- 3 Afferents end directly on Aα neurones supplying same ms or ascends to cerebellum & CC to inform CNS about:
m. length or rate of changing of m. length.

4 Dynamic receptors (BAG) discharge less rapid with sustained stretch.
Excitation of the spindle receptors:

- a Stretch of the whole muscle i.e intrafusal & extrafusal m. fibres.
- b Cont. of intrafusal m.f by ++ γ eff. discharge $\rightarrow$ stretch of central part.
- c Maximal stimulation: Both a + b.

<u>Two TYPES of stretch reflex</u>		<u>SR</u>
	<u>Dynamic SR i.e T jerk</u>	<u>Static SR i.e M. tone</u>
<u>Stimulus</u>	Sudden <u>quick</u> stretch	Slow <u>sustained</u> stretch
<u>Receptor</u>	Nuclear <u>BAG</u>	Nuclear <u>CHAIN</u> & <u>BAG</u>
<u>Afferent</u>	<u>Primary</u> endings	<u>Secondary</u> & <u>prim.</u> endings
<u>Centre</u>	<u>All</u> α motor neurones	<u>Some</u> α motor neurones
<u>Efferent</u>	<u>Alpha</u> fibres	<u>Alpha</u> fibres
<u>Response</u>	Sudden <u>cont</u> followed by sudden <u>relax</u> of extrafusal.	<u>Continuous</u> contraction of alternating extrafusal m. fs.

Effects of gamma efferent discharge: 2

1 Changing sensitivity of spindles to stretch:

Dynamic $\gamma \rightarrow ++$ " to change in rate of " i.e dynamic response

Static $\gamma \rightarrow ++$ " to steady maintained " i.e static response

2 Prevention of unloading « Alpha - Gamma linkage »

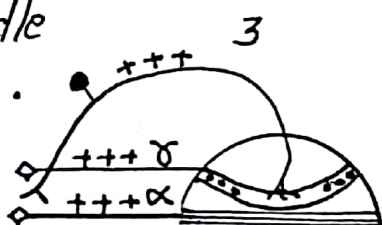
Unloading = $\ominus$ m. spindle firing during m. contraction CNS ②
so, CNS is not informed about m. shortening

Thus, $++\gamma$ eff. discharge with $++\alpha$ eff. discharge allows shortening of extrafusal m.f. without shortening of intrafusal m.f. This keeps CNS informed about changes in muscle length

Physiological significance of SR i.e. m. spindle

1] Helps to maintain muscle length. (discuss).

2] Controls of voluntary movements:



a Servo-assist function during m. contraction.

During vol. contraction, impulses from higher centres stimulate α motor neurones $\rightarrow$ direct cont. of extrafusal m.f.s.

γ motor neurone $\rightarrow$ reflex cont. of extrafusal m.f.s.

So, α - γ linkage potentiates vol. contraction.

b Damping function smoothen muscle contraction



During vol. cont., Δ tr sends irregular signals to Δ HCs $\rightarrow$ jerky mov. SR prevents oscillation & smoothens muscle contraction.

Prove: Deafferentiation i.e. no SR $\rightarrow$ jerky muscle contraction

3] Muscle tone i.e. static SR is the basis of muscle tone

• Definition Reflex subtetanic alternating continuous cont. of sk. m.f.s

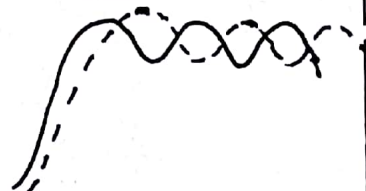
• During rest, m. spindle is continuously stretched because:

a m. length < distance between origin & insertion.

b Force of gravity, i.e. m. tone is more in antigravity muscles

- Flexors of UL
- extensors of LL, back & neck
- elevator of lower jaw.

c continuous γ efferent discharge



• Skeletal muscle tone is maintained without fatigue because:

a subtetanic b alternating c slow (red) m.f.s cont.

• Functions of skeletal muscle tone:

a Maintains body posture b Maintains body temp.

c Background for vol. movements d Helps venous & lymphatic return

Supraspinal centres controlling γ motor neurones & stretch reflex

Facilitatory centers	Inhibitory centres
1 Primary motor area (4) 2 Neo cerebellum 3 Facilitatory reticular formation i.e. pontine reticular nuclei 4 Vestibular nuclei (VN)	1 Cortical suppressor area (4S) 2 Paleo cerebellum 3 Inhibitory reticular formation Medullary reticular nuclei 4 Red nucleus (RN) 5 Basal ganglia
Send impulses via <u>Facilitatory R.F.</u> along <u>Ventral reticulospinal tr.</u> <u>Facilitatory R.F. has intrinsic activity</u> VN sends via <u>Vestibulospinal tr.</u> Act on γ motor neurones <u>reflexly</u>	Send impulses via <u>Inhibitory R.F.</u> along <u>Lateral reticulospinal tr.</u> <u>Inhibitory R.F. has no intrinsic activity</u> RN sends via <u>Rubro-spinal tr.</u> Act on γ motor neurones <u>reflexly</u>

Note Vestibulospinal tr. also stimulates directly alpha motor neurones.

Experimental proof : Decerebrate Rigidity.

Lesion : Animal TS in brain stem between pons & medulla.
Man Internal capsule.

Mech. : lesion removes inhibitory effect of supraspinal center leaving facilitatory effect of facilitatory R.F. & vestibular nuclei

Effects : Rigidity in antigravity ms i.e. extended limbs, neck and limbs
" is of reflex (γ) nature elevated tail

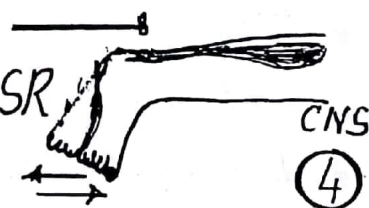
Proof Rigidity disappears by cutting dorsal roots of sp. nerve

Clinical application of SR :

1 Tendon Jerk (Deep reflexes) Dynamic SR

Method : Sudden tapping of tendon

Examples : Ankle, knee, biceps and triceps jerks



2] Muscle tone :

Method Test degree of resistance by flexing & extending limbs

	<u>Areflexia</u> <u>Atonia</u>	<u>Hyperreflexia</u> <u>Hypertonia</u>	<u>Hyporeflexia</u> <u>Hypotonia</u>
Cause: Interruption of	Reflex arc	Inhibitory imp.	Facilitating imp.
e.g	Peripheral neuritis Tabes dorsalis Polio myelitis	UMNL Anxiety Hyperthyroidism	Neocerebellar lesion Sleep Myxedema

Inverse stretch reflex : Autogenic inhibition

Stimulus ++ tension in muscle or tendon e.g. marked stretch.

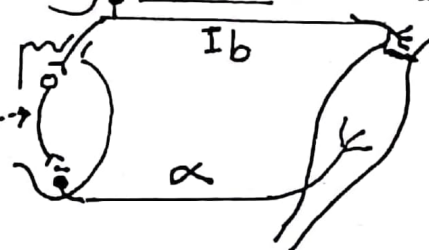
Receptor Golgi tendon organ.

Afferent I_b (myelinated).

Centre Bisynaptic . one inhibitory interneurone

Efferent α alpha.

Response Relaxation.



Aim To maintain muscle TENSION constant

Findings associated with hypertonia due to UMNL :

1 Lengthening reaction i.e. 'Clasp Knife Rigidity'

If UL. is passively flexed, the extensor ms are lengthened
1st m. tone (resistance) due to activation of stretch reflex.

then m tone (resistance) suddenly disappears due to inverse stretch R.

2 Clonus

Def. regular rhythmic (repeated) cont. of muscles

when subjected to sudden maintained stretch.

e.g Ankle clonus when " " dorsiflexion of foot

Mechanism : Alternating stretch reflex & inverse stretch R.

CNS

Polysynaptic Reflexes

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Prolonged response (after discharge) in polysynaptic R. is due to :

- Parallel chain circuits & - reverberating circuits

1) Superficial reflexes i.e. receptors in the skin

1) Flexor withdrawal reflex :

Stimulus : Injurious (painful)

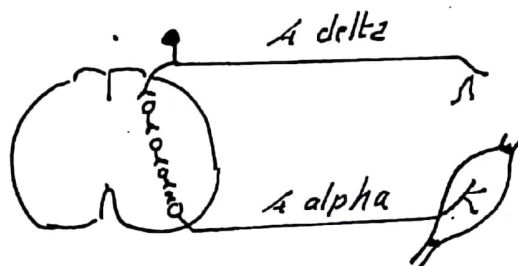
Receptors : Free n. ending

Afferent : Δ delta (mainly)

Center : Polysynaptic

Efferent : Δ alpha

Response : Flexion & withdrawal of limb away from injurious S.



Properties of Flexor withdrawal reflex

a Reciprocal innervation cont. of flexors & relax. of extensors

b Crossed extensor reflex Stronger stimulus produces extension of the opposite limb to support body weight

2) Abdominal reflex

Stimulus : Stroking the skin of abdomen with blunt object.

Response : Contraction of abdominal muscles

Centre : Upper abdom. 7-10 th. Lower abdom 10-12 th.



3) Cremasteric reflex

Stimulus : Scratching skin of upper inner aspect of thigh.

Response : Cont. of cremasteric m. $\rightarrow$ elevation of testis

Centre : 1st & 2nd lumbar segments



4) Planter reflex

Stimulus : Scratching outer aspect of sole by a blunt object

Response : Planter flexion of all toes

Centre : 1st & 2nd sacral segments

CNS

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5) +ve supporting reaction no reciprocal innervation

Stimulus Deep pressure on sole of foot

Response Cont. of flexors & extensors → limb is rigid column.

6) Scratch reflex

Stimulus Tickling or itching e.g. moving insect.

Response Too & fro scratching movements.

7) Local skin reflexes Local skin cold → VC

Local skin heat → VD and sweating



B) Visceral reflexes e.g. Micturition, defecation & erection.

Complete TS of sp. cd. causes trauma or tumours

1 Above cervical 5 : respiratory failure and death.

2 Below cervical 5 : diaphragmatic resp. and quadriplegia

3 Mid thoracic : normal respiration and paraplegia

Permanent loss below the level of :

a All sensations b All vol. movements (fns never regenerate)

Reflexes pass in two stages : sp. shock & recovery of reflexes

a) Stage of sp. shock Loss of all reflexes below lesion level.

① Stretch R. : Atonia (Flaccidity) Loss of tendon jerks.

② Loss of flexor withdrawal reflex :

3 Loss of visceral reflexes : Micturition, defecation & erection.

4 Micturition reflex Retention with overflow.

Urine retention → ++ vesical P > tone of int. sphincter → urine dribbles

5 Venous return is reduced → lower limbs become cold & blue.

6 ABP the higher the lesion level, the lower is the ABP

Explanation Symp. LHCs are separated from VC centre in medulla _{CNS}

→ VD → immediate fall in ABP

⑦

⑦ Bed sores at p. points due to -- bl. flow e.g. over bony prominences

Decubitus ulcers occur if pt mobilization & cleaning are neglected :

③ Muscle wasting occurs

Cause of sp. shock : Sudden withdrawal of supraspinal facilitation.

→ Hyperpolarization (2-6 mV higher than RMP) of sp. motor neurones

Duration of sp. shock : Depends on degree of brain development i.e. encephalization (dominance of CC on sp. cd.) Minutes in frogs 2-8 W in man.

Care of pt with complete TS of sp. cd. reduced mortality from 80 to 6%.

1 Catheterization to prevent urine stasis 2 Rectal enema

3 Frequent mobilization to „ bed sores 4 Antibiotics to prevent infect.

5 Attention to nutrition and fluid balance

b) Stage of recovery of reflexes

① Stretch R : 1st reflex to recover 1st in flexors → paraplegic in flexion

② Flexor withdrawal R : Following planter stim → +ve Babinski sign i.e. dorsiflexion of big toe. Then, flexion of foot, knee and hip.

③ Deep reflexes jerk then Ankle jerk.

④ Mass reflex

- Stimulus Scratching skin of lower limb or abdominal wall.

- Response a Flexor withdrawal of LL & contraction of abdominal wall.

- b Automatic bladder & rectum c Sweating below the lesion.

- d ABP rises

- Mechanism Increased excitability of sp. cd neurones

- Importance It gives paraplegic pt a degree of bladder & bowel control.

⑤ Autonomic reflexes

- a Automatic bladder and rectum.

- b ABP gradually rises to normal (LHCs discharge by themselves)

- Pt has orthostatic hypotension (No medullary vasomotor control)

- c Return of tone of arterioles & venules & improved limb circulation

d Coitus reflex: Genital manipulation in $\sigma \rightarrow$ erection & ejaculation

⑥ Six months after cord TS Mass reflex disappears.

Tone of extensors > Flexors $\rightarrow$ paraplegia in extension
+ve supporting reaction $\rightarrow$ Pt can stand without support.

Recovery of reflexes is due to denervation hypersensitivity
and increased collaterals from existing neurons.

Motor functions of brain stem.

Brain stem contains centres for CVS, resp., GIT, eye movements, body posture and complex reflex movements.

Reticular formation of brain stem large structure in core of B. stem

Two types of neurones: Sensory & Motor neurones (larger, less in number & receive impulses from sensory neurones)

Axons of motor neurons are divided into: discuss page 4

1 Pontine facilitatory RF

Dorso lateral part of pons

It discharges spontaneously

2 Medullary inhibitory RF

Ventro medial part of medulla

It has no intrinsic activity

Facilitatory RF gives ascending branches RAS reticular activating system

RAS passes to non specific thalamic nuclei, basal ganglia to CC.

RAS controls brain activity as consciousness and alertness.

Equilibrium and Vestibular Apparatus

Bony labyrinth in temporal bone = vestibule, bony cochlea & 3 SCCs.

Membranous „ = Auditory labyrinth i.e. membranous cochlea and

Non-auditory labyrinth i.e. utricle, saccule and 3 SCC

It contains endolymph (rich in K^+) & surrounded by perilymph (rich in Na^+)

Nerve supply vestibular division of vestibulo-cochlear nerve ^{CNS}

Semicircular canals SCCs

3 SCCs perpendicular to each other 3 planes of space

⑨

3 SCCs are ant. vertical, post. vertical & horizontal (external).

Receptors of SCCs : Crista Ampullaris :

located in ampulla of each membranous SCC

consists of hair cells, each has 30-150 rod shaped processes.

One kinocilium on one side (largest process with clubbed end).

Other are stereocilia that increase in height towards the kinocilium.

Processes project in endolymph & embedded in cupula (gelatinous partition)

Bases of hair cells in perilymph in contact with afferent nerve fibres

Tip links at processes function as mech. sensitive cation (K^+ & Ca^{++}) channels

Function detects ANGULAR acceleration.

RMP of hair cells = -60 mV

When stereocilia are pushed towards kinocilium $\rightarrow$ open cation channels $\rightarrow$ Depol. $-50\text{ mV} \rightarrow ++$ discharge of APs & vice versa.

Depol. releases glutamine $\rightarrow$ depol. of neighboring afferent neurones

Events occurring in response to horizontal rotational acceleration 5

- At rest, SCCs on both sides discharge equally i.e 200 impulses/sec.
- When the head begins to rotate to right in horizontal plane endolymph by inertia flows to left $\rightarrow$ deflection of both cupulae.
 - Rt cupula is bent towards utricle i.e stereocilia are pushed towards kinocilium $\rightarrow ++$ frequency of impulses in Rt horizontal SCC.
 - Lt cupula is bent away from utricle $\rightarrow --$ frequency of impulses

Result Person has a true feeling of rotation to the right.

- With continuation of rotation with constant speed, inertia of endolymph is overcome $\rightarrow$ both cupulae regain their resting position $\rightarrow$ no sense of rotation
- When rotation is stopped, endolymph continues to flow to Rt by momentum $\rightarrow$ cupulae are deflected in opposite direction i.e Lt cupula towards utricle $\rightarrow$ false sensation of rotation in opposite, i.e to Lt called vertigo (10)

- In few second, endolymph stops moving & vertigo disappears.

Effects of stimulation of SCCs :

1] Sensation of rotation : At beginning of rotation, in same direction.

2] Vertigo :

- Definition false sensation of rotation at rest.
- Mechanism Movement of endolymph e.g by momentum with stop of rotation
- Causes a Stoppage of rotation
b SCCs or vestibular pathway irritation by alcohol or streptomycin.
c Motion sickness due to excess & repeated labyrinthine stimulation
d Menier's disease due to enlargement of SCCs & ++ endolymph pressure

3] Post pointing test of Barany (post-rotatory reactions)

Cortical voluntary movements of limbs & body compensatory to vertigo.

It results in post pointing or even fall on opposite side.

4] Autonomic changes : Nausea, vomiting, pallor & sweating
Hypotension, bradycardia & ++ rate of respiration.

5] Muscle tone changes : (Maintenance of balance)

increased muscle tone on ipsilateral side of rotation.

decreased muscle tone on contralateral side of rotation.

6] Nystagmus :

- Definition Series of jerky eye movements or too stro eye movement
- Components a Slow component opposite to direction of rotation
(Vestibulo-ocular reflex = VOR)
b Fast component in direction of rotation at beginning of "
- Time At the start and at the end of rotation
- Aim To fix objects in visual field during rotation.
- Types Horizontal , Vertical or Rotatory CNS
If the head rotates horizontal sideward forward. (11)

• Causes of nystagmus

Physiological nystagmus

- 1 With head rotation (VOR)
initiated by SCCs impulses
Can occur in blind person
- 2 Optokinetic nystagmus
initiated by visual impulses
e.g. when person looking from
window of moving train

Pathological nystagmus

- 1 Neocerebellar syndrome
- 2 Menier's disease
- 3 Searching nystagmus in defective vision
Focus image on healthy part of retina
- 4 SCCs or vestibular nerve pathway
irritation or lesion

Nervous connections of SCCs (vestibular apparatus)

Vestibular nerve → Vestib. ganglion (19,000 neurons) — Vestibular nucleus then.

- 1 Reticular formation: centre of autonomic changes.
- 2 Vestibulo-spinal & reticulo-spinal trs → changes in muscle tone.
- 3 Medial longitudinal bundles on both sides to 3rd, 4th & 6th cranial nuclei
→ extraocular ms → slow components of nystagmus.
Fast component centre is midbrain & sends impulses also viz MLB
- 4 Cerebellum Flocculonodular lobe & dentate nucleus to fastigial nucleus
to opposite thalamus to equilibrium centre in superior temporal gyrus
→ vertigo. Some fibres pass directly from receptors to cerebellum.

Methods of stimulation of SCCs

- 1 Rotatory method viz rotating chair
Stimulates two canals in two ears adjusted to lie on horizontal plane.
- 2 Caloric method viz washing ear by warm (43°C) or cold (20°C) water.
to produce convection current in endolymph.
Stimulates one canal in one ear adjusted to lie on vertical plane
e.g. tilting head 60° backwards orients horizontal canal in " "
- 3 Electrical method viz stim. vestibular nerve by galvanic current (2-5 mA)
Stimulates three canals in one ear.

Utricle and Saccule (U.S.S)

Receptors : Otolith organ (macula)

Site On floor of utricle In the wall of saccule

Structure a Sustentacular cells

b Hair cells oriented in all directions

Surrounded by otolith membrane embedded in it crystals of Ca carbonate (otolith or otoconia or ear dust) 3-19 μ s denser than endolymph

In utricle, macula lies in horizontal plane & hair cells pointing upwards

In saccule, " " in vertical " & " " " laterally

N.B Maculae don't operate when head is in vertical position

Functions of utricle & saccule:

A Maintenance of static equilibrium

- 1 With head vertical, Rt & Lt U. are equally stim. i.e no m/equilibrium sensed
- 2 If head tilted to one side, ++ discharge on that side & -- discharge on other side so, sensation of m/equilibrium is created.

Equilibration centres in brain stem & cerebellum send corrective impulses.

B Detection of linear acceleration

- 1 Horizontal e.g acceleration of vehicle or running. Utricles send corrective impulses to ms $\rightarrow$ Forward leaning to prevent falling.
- 2 Vertical acceleration is sensed by saccules

Posture and postural reflexes

Equilibrium is maintained by moving centre of gravity or area of support (feet) so that centre of gravity lies within area of support.

Postural reflexes are achieved by changing muscle tone (stretch reflex)

Function of postural reflexes:

- 1 Maintain upright position & restore it if it is lost
- 2 Provide stable postural background for voluntary activity.

CNS (13)

Types of postural reflexes (PR)

1 Static: involve sustained cont. 2 Phasic (dynamic) involve transient cont.

According to their centres PR are classified into:

1] Spinal PR: Stretch R, Crossed extensor R & +ve supporting reaction.

2] Medullary statotonic PR

PR	Stimulus	Receptors	Respond.
a Tonic labyrinthine	Gravity	Otolith organ	Cont. of limb extensor muscles
b Tonic neck reflexes	Head turned 1 to side 2 up 3 down	Neck proprioceptors " "	Extension of limbs side of head turn Fore legs extend & Hind legs Flex Fore legs Flex & Hind legs extend

3] Midbrain righting PR

a Labyrinthine righting reflex on head	Gravity	Otolith organ	Head is kept with both eyes on horizontal plane
b Body righting reflex on head	Pressure on one side of body	Exteroceptors (body proprioceptors)	"
c Neck righting reflex on body	Stretch of neck ms	It. spindles of neck ms	Righting of body position
d Body righting reflex on body	Pressure on one side of body	Exteroceptors (body proprioceptors)	"

4] Cortical PR centre is cerebral cortex

a Optical righting reflex	Visual signals	Rods & cones	Righting of head position & body position
b Placing reactions	Viewing or touching surface	Rods, cones & exteroceptors	Placing foot on surface to support body position
c Hopping reactions	Lat. displacement while standing	Muscle spindles	Hops & maintains limbs in position to support body position CNS (14)

- Notes 1 Equilibrium centre: superior temporal gyrus of cortex
- 2 Receptors: eye, vestibular apparatus, body & neck proprioceptors
- 3 Vestibule & neck proprioceptors function in opposite directions
- a If a person bends his neck: Impulses from neck proprioceptors balance vestibular information $\rightarrow$ person feels head in bent but no mal equilibrium.
- If the entire body leans in one direction No impulses from neck proprioceptors to balance vestibular information $\rightarrow$ mal equilibrium is felt
- b If head of medullary animal is bending forwards: vestibule tends to extend fore limbs while neck proprioceptors tend to flex them.
- 4 If vestibule is destroyed, equilibrium is maintained by eyes.
- 5 During diving in water, body proprioceptors & eye receptors are ineffective. Vestibular receptors are the only functional receptors.
- 6 With closed eyes, a person keeps his equilibrium with proprioceptors
So, in tabes dorsalis, closure of eyes $\rightarrow$ falling +ve Romberg's sign

Motor Cortex

Normal voluntary mov. needs the integrity of 2 neurones:

- 1 Upper motor neurone UMN: From CC and brain stem to AHCs and cranial nuclei i.e. pyramidal & extrapyramidal tracts
- 2 Lower motor neurone LMN From AHCs & motor cranial nuclei to muscle

Motor areas of cerebral cortex includes

- 1 Primary motor area (Area 4)
- 2 Premotor area
- 3 Supplemental motor area
- 4 Somatic sensory area I & II

Primary motor area (motor area 4)

Site: precentral gyrus

It is highly excitable due to presence of Betz cells

Representation 1 Crossed and unilateral, except the upper part of face

CNS

(15)

- which is represented in both cerebral hemispheres
- 2 Inverted
 - 3 Proportional to complexity of skilled mov. e.g. speech & hand ms
- Functions
- 1 Controls Fine discrete skilled mov. of distal parts e.g. fingers
 - 2 Facilitatory to stretch reflex and muscle tone

Supplementary motor area small area

Site in front of motor area 4 & above premotor area

Function Projects to motor cortex & involves in programming motor sequence.

Blood flow to this area increases with complex movements

Premotor cortex (motor association area or area 6 and 8)

Site in front of prim. motor area, above Sylvian Fissure

Less excitable than area 4 no Betz cells

Body representation inverted

Function Complex coordinated movement by sending signals to area 4 to excite multiple group of muscles

Premotor area contains 4 areas from below upwards:

[a] Broca's area: word formation or speech area

Site Ant. to lower end of motor area 4 in dominant hemisphere

Function projects signals to motor area 4 which lead to vocalization

Damage Motor aphasia Vocabulary is limited to few words e.g. yes/no

[b] Voluntary eye movement area: Above Broca's area

Function connected to occipital visual area to control eye movement

Damage no eye movements towards different objects.

[c] Head rotation area: Above eye movement area.

Function connected to eye mov. area & move head towards diff. objects

[d] Hand skills area

Site Ant. to primary motor area for hands & fingers

Function Coordinated skilled hand movements

CNS

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Damage Motor apraxia uncoordinated hand movement due to loss of memory to complex skilled hand movement.

Connection of motor cortex

A Afferent Fibres

- 1 Subcortical Fibres from | somatic sensory areas
corresponding area of opposite motor cortex.
- 2 Thalamic fibres including | sensory fibres via ventrobasal complex of thalamus
basal ganglia & cerebellum via
ventroant. & ventrolat. nuclei of thalamus
non-specific thalamic nuclei

B Efferent Fibres Δ trs & extra Δ trs i.e UMN to LMN Pyramidal system 3 tracts

1 Cortico-spinal tr

A In cerebral hemisphere

- 1 Cortex one million fibres (30% from prim. motor area, 30% from premotor area & 40% from somatic sensory areas (post. to central sulcus)
3% of fibres large myelinated fibres, derived from Δ betz cells
- 2 Subcortical region fibres form corona radiata
- 3 Internal capsule $\rightarrow$ pass in genu & posterior limb.

B In brain stem: Fibres pass

- 1 Midbrain: in cerebral peduncle.
- 2 Pons: between transverse fibres.
- 3 Medulla: in pyramids.

- 85% cross in medulla $\rightarrow$ crossed or lat. corticospinal tr.
- 15% cross in neck & upper thorax $\rightarrow$ direct or ant. corticospinal tr
- few fibres of ant. corticosp tr don't cross & provide bilateral innervation to upper face, respiratory & abdominal muscles

N.B Lat. corticosp. tr controls distal muscles of limbs & hands CNS 17
Ant. corticosp. tr controls axial (trunk & proximal limb) muscles

<u>Corticobulbar tract</u>		<u>Cortico-nuclear tract</u>
prim. & premotor areas at brain stem	Arise from	area 8
5, 7, 9, 10, 11 & 12th in pons & medulla	Cross	at brain stem
control muscles of head	Synapse with motor nuclei	3rd, 4th, 6th (extraocular ms) on both sides
	Function	deviation of 2 eyes to opposite side

Functions of pyramidal system:

- 1 Complex fine skilled vol. movements of finger, toes & face.
- 2 Facilitatory to muscle tone (evidence: section $\rightarrow$ Hypotonia)

II Extrapyramidal system: other motor tracts

Arise from: Premotor area, BG, RF, red nuclei & vestibular nuclei.

Extra Δ ts: Rubro, Reticulo, Tecto, Vestibulo & Olivospinal trs to 4HCs

Functions 1 Gross movements involving group of ms required for fixation
2 Some fibres are facilitatory, others are inhibitory to muscle tone.

<u>Upper Motor Neurone Lesion (UMNL)</u>	<u>Lower Motor Neurone Lesion (LMNL)</u>
<u>Cause</u> Cerebrovascular accidents as strokes due to hge, thrombosis in post limb of internal capsule $\rightarrow$ Lesion of Δ & extra Δ trs	<u>Cause</u> 1 Lesion of neurone e.g. poliomyelitis 2 Lesion of nerves DM & alcoholism 3 Lesion of N-M junction: M. gravis
Paralysis usually on <u>opposite side</u> & widespread <u>contralateral hemiplegia</u> <u>Poor recovery</u> .	Paralysis always on <u>same side</u> & usually <u>localised</u> <u>Recovery</u> may occur.
<u>Characters of paralysed muscles</u> 1 <u>Hypertonia</u> Spastic paralysis Clasp knife type i.e. resistance to passive movement suddenly gives way 2 <u>Exaggerated deep reflexes</u> (tendon jerks) 3 <u>Clonus</u> is present	<u>Characters of paralysed muscles</u> 1 <u>Hypotonia</u> or atonia i.e. Flaccid paralysis Due to interruption of Stretch R. are 2 <u>Absent deep R.</u> (tendon jerks) 3 <u>Clonus</u> is absent

- 4] Loss of superficial reflexes
Due to loss of supraspinal facilitation
Planter reflex: +ve Babinski sign
- 5] No significant ms wasting
due to hypertonia & hyperreflexia
- 6] No Fasciculations
- 7] Electrical excitability is normal
Faradic: tetanic contractions
Galvanic: CCC > ACC
Chronaxie: normal

- 4] Loss of superficial reflexes
- 5] Marked ms wasting (disuse atrophy)
due to atonia & areflexia
- 6] Presence of fasciculation due to
Pathological discharge of sp. neurones
- 7] Reaction of degeneration.
Faradic: no response
Galvanic: ACC > CCC
Chronaxie: Prolonged.

CCC = cathode closing contraction. ACC = anode closing contraction

N.B Babinski's sign is abnormal response to planter reflex
i.e dorsiflexion of big toe & fanning of other four toes
It is a primitive response following injury of Δ tr.

Other causes of Babinski's sign :

- 1 Deep sleep, deep coma and during anaesthesia in adults
- 2 In infant below one year; due to immaturity of pyramidal tr.

Effect of lesion of internal capsule "posterior limb" 4 H.

Causes thrombosis or hge in lenticular artery

- 1 Hemiplegia due to UMNL Discuss table
- 2 Hemianesthesia on opposite side due to injury of sensory radiation
- 3 Hemianopia Crossed homonymous hemianopia due to "of optic"
- 4 Steering in both ears is slightly affected due to "of auditory"
But no deafness; as each ear is bilaterally represented in both auditory cortex.

Basal ganglia

- 1 Caudate
- 2 Putamen
- 3 Globus pallidus

} striatum

} lenticular n.

5 related structures

2 functionally related nuclei

1 Subthalamic nucleus

2 Substantia nigra

Neuronal connection of basal ganglia (BG)

BG are connected to each other & with other areas of n. system.

1) Cortical connections

2 main circuits

Caudate circuit

- Cortical association areas (motor, sensory, visual & auditory)
- To CAUDATE nucleus.

Putamen circuit

Premotor & supplementary motor areas of motor cortex.
To PUTAMEN.

- To internal globus pallidus to
- Thalamus ventroanterior or ventrolateral nuclei (VATN or VLTN)
- To prefrontal, premotor & supplementary areas concerned with planning & sequences of pattern

To primary motor cortex concerned with patterns of movement

N.B Fibres from CC to neostriatum secrete acetylcholine (excitatory).

Fibres from neostriatum to substantia nigra (SN) secrete GABA (inhibitory)

Fibres from SN to neostriatum secrete dopamine (inhibitory).

2) Efferent pathway from BG

Globus pallidus is the chief efferent pathway of BG. It sends impulses to subthalamic nuclei & SN then to reticular formation (RF). RF sends via extrapyramidal trs (reticulospinal, vestibulospinal & rubrospinal) to motor neurones of sp. cord.

Balance between excitatory & inhibitory transmitter is needed for BG function.

Functions of BG Important role in motor control

1 Caudate nucleus with CC helps in:

- a planning sequences of movements to achieve a complex goal
 - b control timing (rapid & slow) and scale i.e spatial dimensions
e.g small or large letters
 - 2 Putamen circuit with corticospinal system execute subconscious learned pattern of movement e.g writing letters, cutting with scissors & some aspect of vocalisation.
 - 3 Initiation & regulation of gross intentional movements
e.g swinging of arms while walking and facial expressions
 - 4 Globus pallidus is responsible for posture taken to perform particular voluntary movement e.g it locks arm in specific position to facilitate fine movements of the hand
 - 5 Basal ganglia is mainly inhibitory to muscle tone
- Metabolic consideration
- 1 BG have a high O₂ consumption
 - 2 SN has high content of copper. In Wilson's disease, ceruloplasmin is low → chronic Cu intoxication & degeneration of lenticular nucleus
- Diseases of BG Involuntary mov. + change in M. tone look table
- Treatment of Parkinsonism

- 1 L-dopa (levodopa), unlike dopamine, can cross blood brain barrier.
- 2 Anticholinergic drugs decrease cholinergic influence
- 3 Surgical: cutting in internal segment of globus pallidus (pallidotomy) or in subthalamus nuclei to restore output balance toward normal.
- 4 Implantation of dopamine secreting tissue in or near basal ganglia

Cerebellum

It is connected to brain stem by superior, middle & inferior cerebellar pedunc.
It is divided into 3 areas with different functions

Lobe	Connected to	Function
1 <u>Archi</u> (vestibulo) cerebellum i.e Flocculo-nodular lobe	Vestibular nuclei	Equilibrium (21)

Diseases	Chorea	Parkinsonism (Paralysis Agitans)	Athetosis	Ballism
Lesion	Caudate & Putamen	Substantia nigra	G. pallidus	Subthalamus
Pathology	Hereditary disease Stuntington's chorea Rheumatic fever in children	1 Cerebral atherosclerosis → -- dopamine receptors 2 D ₂ Dopamine receptor blockers e.g. phenothiazine 3 Head trauma	Wilson's disease → decreased ceruloplasmin	Vascular
Mechanism	Degeneration of neurones secreting 1 GABA (inhibitory) → no inhibition to SN. → ++ dopamine secretion → (+) of neostriatum 2 Acetylcholine (excitatory)	Loss of dopaminergic inhibition of putamen leading to: Imbalance between inhibition and excitation → <u>excitation</u>		
Characters	DANCING movement Spontaneous & rapid HYPOTONIA Pendular knee jerk	3 x 3 1 <u>STATIC TREMORS</u> rhythmic alternating • During rest • Disappear by movement • Site hand (pill rolling) & mandible 2 <u>RIGIDITY</u> • More in <u>flexors</u> → Flexion attitude • Lead-pipe i.e. continuous or Cogwheel i.e. series of catches • Cause ++ discharge in α & β AHG 3 <u>AKINESIA</u> difficult initiating voluntary spontaneous mov. • Face: mask (no expression). • Speech: monotonous. • Gait: shuffling legs short step arms no swinging	<u>WORM LIKE</u> continuous & slow in arm & hand & head & neck <u>HYPERTONIA</u>	<u>FLAILING</u> intense violent <u>HYPOTONIA</u>
NO sensory disorder	Strictly motor i.e. disorder			

Lobe	Connection	Function
2 <u>Paleo (spino) cerebellum</u> : i.e midline area (vermis) and 2-3 cm wide in both ant & post lobes	<u>AHCs</u> & <u>CC</u>	Coordination of movements Inhibition of muscle tone
3 <u>Neo (cerebro) cerebellum</u> : i.e lat parts of cerebellar hemisphere	<u>Cerebral</u> <u>Cortex</u>	Planning & programming of movements. Facilitatory to muscle tone

- Ant. lobe: body is represented upside down.
- Post. lobe: body is represented erect
- Lateral zones: no topographic representation as these areas have a different function i.e planning & programming of vol. movem.

Cerebellar connections

- Afferent Fibres (climbing and mossy fibres) end in cerebellar cortex
middle layer of Purkinje cell (cortex is grey matter. formed of 3 layers;
superficial molecular, middle Purkinje & deep granular)
Axons of Purkinje cells carry efferent impulses to cerebellar nuclei
i.e dentate, interpositus & fastigial nuclei from which impulses pass
to various areas of brain e.g brain stem & thalamus.

Cerebellar peduncle	Afferent i.e cerebellar tract
1 <u>Superior</u>	<u>Ventral spino</u> cerebellar tr
2 <u>Middle</u>	<u>Cortico-ponto</u> cerebellar tr
3 <u>Inferior</u>	<u>Dorsal spino, vestibulo solivo</u> cerebellar trs

• Efferent Fibres

Cerebellar peduncle	Efferent
1 <u>Superior</u>	a Dentate thalamo - cortical tr b Dentato-rubro - spinal tr
2 <u>Inferior</u>	a Fibres to reticular formation of <u>pons</u> (23) b Fibres to reticular formation of <u>medulla</u>

Because of double crossing each cerebellar hemisphere acts on ipsilateral muscles; dentato-rubrospinal tr arises from dentate nucleus and passes to red nucleus of opposite side again rubro-spinal tract crosses to the opposite side

Functions of cerebellum

Stim of cerebellum doesn't produce sensations or motor action i.e. silent area

Cerebellum Coordinates motor activity; it is very important in precise execution of Rapid muscular action.

1. Functions in voluntary movement:

1] Servo-comparator Function:

- Cerebellum is informed about:
 - a Signals (order) transmitted from motor cortex to a group of ms via Cortico-Ponto-Fastigial tr to intermediate zone of cerebellum
 - b Performance of ms via spino-cerebellar trs
- Cerebellum compares performance with order
- If not appropriate, cerebellum initiates corrective signals to motor cortex via Fastigial-thalamocortical tr.

2] Braking effect of cerebellum

Brakes stop movements at precise intended point.

Mechanism Cerebellum assesses rate of movements & calculate length of time needed to reach intended point & then sends inhibitory impulses to motor cortex to inhibit agonist and excite antagonist muscles

3] Planning and timing function of cerebellum

Lateral zones of cerebellum plans for next movement at same time present movement is occurring and provides appropriate timing for each movement

Mechanism Lateral cerebellum receives impulses from (24)

cortical association areas (origin of commands) to plan the movements
The cerebellum sends efferent to motor areas of cerebral cortex to initiate movements via corticospinal tr

Lesion of lat. cerebellum: Inability to judge distance moved by a particular part in a given time

B Other Functions

1 Equilibrium

Flocculonodular lobe is connected by feedback circuit with vestibular apparatus. They play import. role in maintenance of equilibrium. Signals from vestibular apparatus to cerebellum to brain stem pass via reticulosp. tr & vestibulosp. tr to maintain equilibrium by changing muscle tone in axial and girdle muscles.

Lesion of Flocculonodular lobe: Disturbance of equilibrium.

2 Muscle tone

- Neocerebellum is facilitatory to stretch reflex. Since its feedback time is longer than stretch reflex, it prolongs the effect.
- Paleocerebellum is inhibitory to stretch reflex e.g. muscle tone.

Cerebellar lesion in human

Neocerebellar syndrome due to damage of deep nuclei or cerebellar cortex. Manifestations occur on SAME SIDE of lesion.

Manifestations

A Ataxia Incoordination of vol. movements ...

In absence of motor lesion i.e. UMN/L or LMN/L.

i.e. error in rate, range, force and direction of movement

Lesion in cortex: Abnormalities gradually disappear compensation occurs.

Lesion in nuclei: Abnormalities are permanent & more generalised

B Other manifestations

- 1 Disturbance of posture and gait Head is tilted to lesion side

Pt has a wide base, unsteady drunken gait, pt may fall to lesion side

- 2 Dysmetria or past pointing Tested by finger-nose test.
- 3 Dysarthria in form of slurred or scanning speech
- 4 Dysdiadochokinesia: inability to perform rapidly alternating opposite movements e.g repeated pronation & supination of hands
- 5 Intention tremor (kinetic tremors) i.e absent at rest & appears during voluntary action. Cause gross corrective action initiated by dysmetria results in overshooting to other side $\rightarrow$ oscillations back & forth.
- 6 Eye ball tremors nystagmus (horizontal) It occurs when Pt tries to fix his gaze to one side. It is due to absence of damping function.
- 7 Decomposition of movements Difficulty in performing actions involving simultaneous motion at more than one joint
It is tested by heel-knee test
- 8 Rebound phenomenon Inability to put on brake i.e to stop movement promptly if it is needed
- 9 Hypotonia on the side of lesion $\rightarrow$ reduced pendular knee jerk.

Control of voluntary movement

- Initiating command motor signals start at cortical association areas.
- Movements are planned in premotor cortex, basal ganglia & lat. cerebellum and coded in terms of movements not individual cont.
- Motor signals are transmitted via Δ trs & extra Δ trs to spinal and cranial motor neurons.

Thalamus Highest subcortical sensory centre

All sensory signals except olfactory relay in thalamus on their way to CC.

Thalamic nuclei are functionally divided into:

- 1 Non-specific thalamic nuclei: Midline & intralaminar nuclei
Receive signals from RAS
Projects to All areas of cerebral cortex

2 Specific thalamic nuclei : postero ventral nuclei, medial & lateral geniculate bodies and anterior thalamic nuclei

Receive Somatosensory, visual & auditory information from sp. cord and brain stem.

Project to specific sensory areas of cerebral cortex

Other thalamic nuclei receive signals from:

- Hypothalamus and relayed them to limbic system.
- Basal ganglia & cerebellum & project them to motor cortex.

Functions of thalamus

- 1 It is relay station for all sensations (except olfaction) to CC.
- 2 It relays motor signal from basal ganglia & cerebellum to motor cortex.
- 3 It relays autonomic & emotional signals to hypothalamus & limbic system
- 4 It plays a role in coding, storing & recalling memories.
- 5 Non-specific thalamic nuclei are concerned with excitability of cortex.

Thalamic syndrome

Cause Vascular thrombosis → damage of posterior thalamic nuclei leaving medial and anterior group of nuclei intact.

Manifestations + Loss of all sensations on the opposite side of body

2 Ataxia: Sensory ataxia (similar to that of tabes dorsalis)

3 2nd hyperalgesia Pain threshold is increased, but when reached pain is prolonged, severe & very unpleasant.

It is due to ++ sensitivity of medial thalamic nuclei to pain impulse

4 Emotional disturbance

Arousal mechanism Wakefulness

Control of consciousness, alert & electric activity is function of RAS

RAS is multisynaptic pathway; located in reticular formation.

Some branches pass directly to CC others via nonspecific thalamic nuclei

Factors affecting activity of RAS

A ++ RAS sensitivity

- 1 Ascending sensory impulses particularly pain & proprioceptive stimuli
- 2 Descending impulses from CC " emotions & vol. movements.
- 3 Epinephrine & norepinephrine

B -- RAS sensitivity

- 1 Impulses from sleep centres in reticular formation
- 2 Lesions of brain stem e.g. vascular, tumors, poisons or hypoxia
- 3 Drugs e.g. barbiturates → hyperpolarization of neurones.

Electrical activity of brain

2 types

Evoked potentials (EP) & spontaneous potential (EEG)

I Evoked cortical potential

Def : Cortical electric activity produced by receptor stimulation.

Recording : Electrodes placed on surface of CC of anaesthetized animal.

Response : 2 positive waves separated by a negative wave.

A Primary evoked potential (EP)

+ve wave then -ve wave (hyperpol)
It has short latency 5-12 msec.

Recorded from specific localised area of CC e.g. hand area if hand is stimulated

B Diffuse secondary response

Large prolonged +ve wave.

Longer latency 20-80 msec due to - slow impulses in multiple synapses RA

Recorded over large wide area of cortex & other brain structures due to stim of non specific thalamic nuclei

In clinical practice

- Evoked responses is recorded over brain & sp. cd with repeated stim. of receptors
- A computerized averaging technique which summates EP & cancels interference
- Somatosensory, auditory & visual stimuli → SSEP, AEP & VEP respectively.
- Especially VEP is useful in detection, localization & follow up of sensory pathway lesion.

11) Electro-encephalogram (EEG)

EEG is record of spontaneous electric activity of brain
Recording electrodes on scalp & multi-channel recorder
 in a calm room at a comfortable temp Subject is in physical & mental rest.
 Waves of variable intensity 0-200 mV & frequency 1-50 Hz

Types of normal EEG waves 4 types

	Alpha	Beta	Theta	Delta
1. Frequency	8-13 Hz	18-30 Hz	4-7 Hz	1-3 Hz
2. Amplitude	50 μ V	20 μ V	100 μ V	100 μ V
3. Age	Adult	Adult	Child	Infant
4. Recorded from	Parieto occipital	Frontal region	Parietal & temporal	—
5. State of person	Physical & mental rest & waken Eyes closed	Intense stim. of CNS e.g. thinking or tension	In adult under emotional stress	In adult during deep sleep

If theta & delta waves occur in waking adult it indicates abnormality.
 Hyperventilation $\Rightarrow$ Frequency of alpha waves becomes theta or delta.

Desynchronization or alpha block: If eyes are opened, synchronous alpha discharge is replaced by faster low irregular beta waves
 also called arousal or alerting response as it correlates with alert state.

Clinical uses of EEG

1) Localizing brain tumors

- Large tumor reduces electric activity in region of tumor.
- Tumor compresses & excite surrounding areas $\rightarrow$ very high voltage.

2) Diagnosis of epilepsy

- Grand mal epilepsy Aura i.e sensory hallucinations followed by generalised tonic convulsions of body (few seconds to 3-4 minutes)
EEG ++ neuronal discharge from all brain areas & RAS $\rightarrow$ high voltage spikes followed by slow waves all over brain
- Petit mal epilepsy 3-30 sec of unconsciousness, with head twitches

e.g. blinking followed by return of consciousness & previous activity
 EEG shows characteristic spike & dome pattern all over brain

3) Diagnosis of sleep disorders

4) Confirmation of brain death (Flat EEG)

SLEEP

A state of loss of consciousness from which proper stimuli produce arousal.

As person falls into sleep different stages are recorded in EEG.

During alert wakefulness, EEG has a high frequency beta waves.

Followed by quiet wakefulness, person is relaxed with eye closed, EEG α waves
 Followed by

A) Slow-wave sleep (non rapid eye movement) Non-REM

Stage 1 Low-voltage and high frequency waves.

Stage 2 Sleep spindles i.e. burst of α like waves 10-14 Hz 50 micro volts

Stage 3 Increased amplitude low frequency waves.

Stage 4 Large waves with maximum slowing.

Deep sleep has rhythmic slow waves indicating marked synchronization.

B) Rapid eye movement sleep REM

Irregular low voltage high frequency waves like beta waves of alert state.

	Non REM	REM
1. occurrence	Most of sleep 80% of sleep time	Episodes 5-30 minutes every 90 min of non REM 20% of sleep time
2. Eye Movement	Eyes deviate up	Rapid eye movement
3. Talking + Walking	Present	Absent
4. Dreams	Present but not remembered (not consolidated in memory)	Dreams which are remembered.
5. Threshold of a waking	Low	High, difficult to a waken
6. Hear rate, respiratory rate, ABP, metabolic rate.	Decreased	Increased
7. Muscle Tone	Hypotonia	Marked hypotonia
8. EEG	Typically theta + delta with sleep spindle	Beta waves irregular, fast, low waves

Distribution of sleep stages

A young adult enters NREM sleep & spends in stages 3 & 4 70-100 min. Sleep then lightens & REM period follows. This cycle is repeated every 90 min. Thus, 4 to 6 REM periods per night. % of REM sleep $\Rightarrow$ in old age.

Mechanism of sleep

[a] Genesis of slow wave sleep by 3 subcortical regions

- 1 Diencephalic sleep zone in post. hypothalamus.
- 2 Medullary synchronizing zone in reticular formation.
- 3 Basal forebrain sleep zones.

Slow-wave sleep is produced by -- serotonin, ++ adenosine & release of PG D₂ in preoptic area of hypothalamus.

[b] Genesis of REM sleep Cholinergic discharge in pontine R. formation

Sleep disorders

- 1 Insomnia insufficient sleep in adult due to anxiety or coffee (analeptics)
- 2 Somnambulism sleep walking More in σ children. Person walks with eyes opened & avoid obstacle but can't remember what he did.
- 3 Narcolepsy irresistible sleep during activities start with REM sleep.
- 4 Sleep apnea Obstruction of airways during sleep awakens the person.
- 5 REM behavior disorder No hypotonia Pt acts out his dreams

Hypothalamus

plays a role in homeostasis

Receives afferents from pre-olfactory area, limbic lobe, amygdala, hippocampus, nonspecific thalamic nuclei, GP & sensory ascending trs

Sends efferents to neurohypophysis, ant. thalamic nuclei, brain stem RF

Functions of hypothalamus

- 1 Autonomic functions Stim. of ant. nuclei produces parasymp effects
Stim. of post dorsomedial & lat. nuclei produces symp effects
- 2 Behavioral function as part of limbic system It is concerned with affective nature of sensation

Stim of certain areas produces reward & others produces punishment sensation

3 Defensive function as a part of limbic system.

Hypoth. is involved in expression of rage and fear

4 Wakefulness and sleep as a part of RAS it initiates & maintains wakefulness

Stim. of ant. hypoth promotes sleep.

5 Relation to cyclic phenomenon. It adjusts circadian rhythm.

6 Neuro-endocrine function

a Control of post. pituitary

Synthesis of ADH & oxytocin

pass along hypoth-hypoph. tr

b Control of ant. pituitary

Releasing of > 9 releasing hormone

viz hypoth-hypoph portal circ

7 Food intake regulation 2 centres

a Feeding centre in Lateral hypothalamus → hunger

b Satiety centre in Ventromedial nucleus of hypoth. → anorexia.

8 Body water regulation

a Thirst centre in supra-optic nucleus of hypoth b. ADH

9 Control of sexual function viz ant. pit through GnRH

10 Temperature regulation

Post. hypoth. controls heat gain viz cutaneous VC & shivering

Ant. hypoth controls heat loss viz cutaneous VD & sweating

Limbic system and behavior

Limbic system consists of :

a Cortical ring on medial side of each hemisphere surrounding Corpus callosum called allo-cortex composed of orbito-frontal area, subcallosal gyrus, cingulate gyrus, parahippocampal gyrus & uncus

b Subcortical nuclei : hypoth, septum, pre olfactory area, thalamus, parts of basal ganglia, hippocampus & amygdala. (32)

Components of limbic system are connected to each other & with neocortex
Limbic cortex circuits have prolonged after discharge → prolonged

emotional responses that continue after stimuli that initiate them

Functions of limbic system

- 1] Olfaction : It has a role in analysis of olfactory signals.
- 2] Sexual behavior is regulated by limbic system & hypoth. Higher cerebral functions, social & psychic factors modify sexual behavior.
- 3] Control of autonomic behavior limbic system produces autonomic changes that accompany emotional states e.g changes in ABP, HR & resp.
- 4] Control of emotions

Fear and rage are closely related instinctive phenomena.

Fear avoidance reaction occurs when animal is threatened.

Rage Fighting reaction occurs " " is threatened & cornered.

External manifestations of Fear : sweating, pupillodilation

tachycardia, dryness of mouth & escape reaction

Fear centre : periventricular nuclei of hypoth. and amygdaloid nuclei

Lesion in amygdala → disappearance of fear.

Rage centre : Lateral hypoth. & amygdala

Normally inhibited by neocortex or ventromedial nuclei of hypoth.

Lesion in neocortex → rage response to minor stimuli

- 5] Motivation Force activating certain behavior to achieve a goal
- Limbic system contains two areas for motivation.

a Area for rewarding sensation

b Area for punishment sens.

In medial band from frontal cortex

In lateral portion of posterior

via hypoth to midbrain tegmentum

hypothalamus.

Rewarding & punishment are important in:

a Motivating behavior to get reward or avoid punishment.

b Learning : Experience that doesn't result in reward or " is not remembered very well. However, opposite experience → more intense cortical response. i.e reinforced (33)

Higher brain function

Learning and memory

Learning: Acquisition of information Memory Storage of informat.

Learning & memory are closely related

Memory is divided into: Explicit which becomes Implicit

1 Declarative = recognition = Explicit

2 Non declarative = reflexive = Implicit

- Conscious recall
- Dependent on hippocampus & medial temporal lobe

• Types

1 Episodic: for events

2 Semantic: for words, rules & language

• Unconscious automatic recall.

• Doesn't involve hippocampus

• It includes Skills & Habits & Priming i.e facilitation of recognition of words or object e.g physio....

• Types Associative & non associative

[a] Non-associative learning Person learns about single stimulus by

1 Habituation Repeated stimulus $\rightarrow$ -- response

Cause Inactivation of Ca^{++} channels in pre-synaptic neurones

2 Sensitization Repeated stimulus $\rightarrow$ ++ response

If coupled one or more time with pleasant or unpleasant stimulus

[b] Associative learning Relation of one stimulus to another

[1] Classic conditioning Pavlov experiment refer to psychology
Repeated pairing of conditioned stimulus CS (bell ringing) that elicits no response with unconditioned, US (food) that produces salivation $\rightarrow$ CS alone will produce salivation.

Characteristics

a Internal inhibition repeated CS without US $\rightarrow$ no response

b External inhibition If disturbed by ext. stimulus $\rightarrow$ no "

c Reinforcement CS is reinforced from time to time by pairing the CS and US, the conditioned reflex can persist indefinitely

[2] Operant conditioning $\neq$ classic Animal is active (performs a task)
US is pleasant or unpleasant & CS is light or other signals

Explicit memory & many forms of implicit memory involve

<u>Immediate (sensory) memory</u>	<u>Short-term memory</u>	<u>Long-term (fixed permanent)</u>
• <u>Few seconds or minutes</u> • <u>Initial stage for memory</u> • <u>Fades or changes to other types</u>	• <u>Few minutes or hours</u> • <u>Characters :</u> 1 <u>Rapid recall</u> 2 <u>Easily displaced</u> 3 <u>Working memory</u>	• <u>Hours to years or life long</u> • <u>Characters :</u> 1 <u>Not instantaneous</u> 2 <u>Resists disruption</u> 3 <u>Develops from other forms</u>
• <u>Molecular base</u> ++ <u>synaptic transmission</u> - <u>Reverbrating circuits</u> - <u>Post tetanic facilitation</u>	• <u>Molecular base</u> <u>Temporary memory base</u> i.e. new <u>facilitated pathways</u> - <u>synaptic sensitization</u> - <u>long term potentiation</u>	• <u>Molecular base</u> <u>Structural change in presynaptic</u> <u>Memory engram</u> ++ <u>number of terminals</u> ++ <u>of transmitter vesicles</u> <u>activation of genes & protein synthesis</u>

Important (death) or repeated Immediate or short term memory is changed to long term
Consolidation of memory trace encoding memory & making it resistant to erase
 It involves hippocampus and its connection.

Centres For memory encoding and storage :

- 1 Implicit memory is encoded in stratum of BG & cerebellar folliculus
- 2 Explicit memory - Short term is encoded in hippocampus
 - Long term (Emotional aspect) in neocortex and amygdaloid nuclei

Clinical links 1 Amnesia (Loss of memory) - Retrograde : events before injury
 - Antrograde : events after injury

- 2 Alzheimer's disease Progressive loss of short term memory
 Followed by loss of cognitive and other brain functions

Cause Degeneration of hippocampal neurones

Speech and language

Highest Function

It involves understanding & expressing ideas whether spoken or written

Speech centres :

- 1 Prim. visual area (Area 17) in occipital lobe : See written words.
- 2 Prim. auditory area (Area 41, 42) in temporal lobe : hear spoken words (35)

Further analysis and interpretation of visual & auditory signals are function of sensory association areas which include

- 3] Visual association area (Area 18, 19): understand meaning of written words
- 4] Auditory association area (Area 22): „ meaning of spoken words or sounds
- 5] General interpretative area Wernicke's area More developed in one hemisphere

Located in superior temporal lobe where somatic, visual & auditory association areas meet

It interprets auditory & visual information to understand or express thoughts
Wernicke's area project to

- 6] Hand skill area (Exner's area) 7 Broca's area (Area 44)

Located in premotor area

Hand movement For coordinated program for Vocalization

Hand area It projects to prim. motor cortex to Face area

Aphasia Abnormalities of language functions

due to injury of „ centres in cerebral cortex.

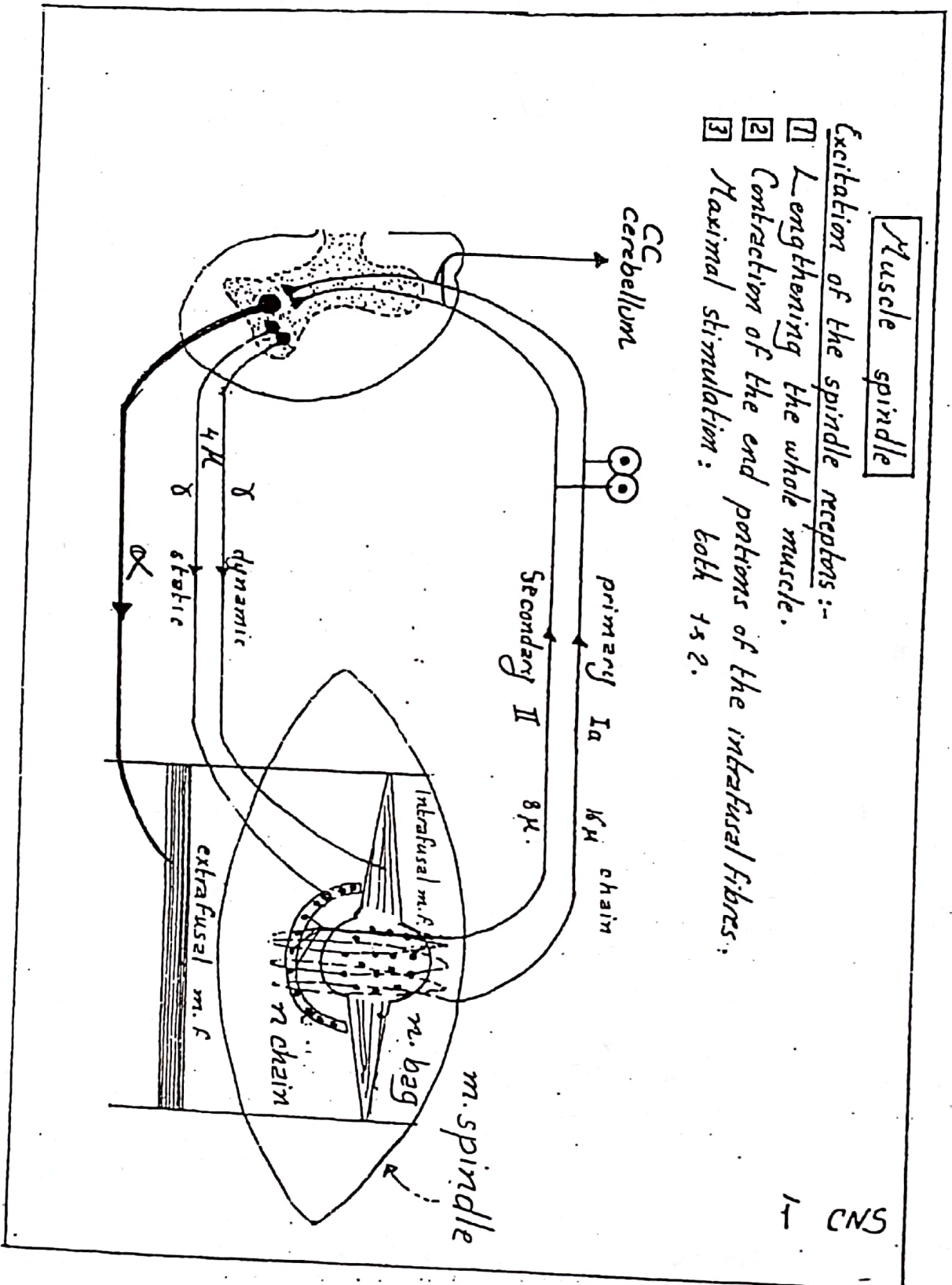
Causes : Usually follows thrombosis or embolism of „ vessels

Type	Area Damaged	Defect
1. <u>Auditory aphasia.</u> = word deafness	<u>Auditory association area</u> in <u>super. temporal gyrus</u>	Person is unable to understand <u>spoken words</u>
2. <u>Visual aphasia</u> = word blindness	<u>Visual association area</u> in <u>occipital lobe</u>	Person is unable to understand <u>written words</u>
3. <u>Wernicke's aphasia.</u> = Fluent aphasia	<u>Wernicke's area</u>	Person is unable to a <u>interpret meaning</u> of spoken or written words b <u>express thoughts</u> into words Talk is excessive & meaningless
4. <u>Broca's aphasia</u> = Non-fluent = Motor aphasia	<u>Broca's area</u>	Person is unable to <u>produce words</u> instead of noises Speech is poorly articulated & slow and with great effort It may be limited to two or three words

Muscle spindle

Excitation of the spindle receptors:-

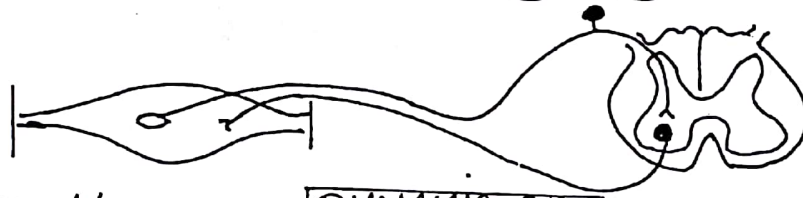
- 1 Lengthening the whole muscle.
- 2 Contraction of the end portions of the intrafusal fibres.
- 3 Maximal stimulation: both 1 & 2.



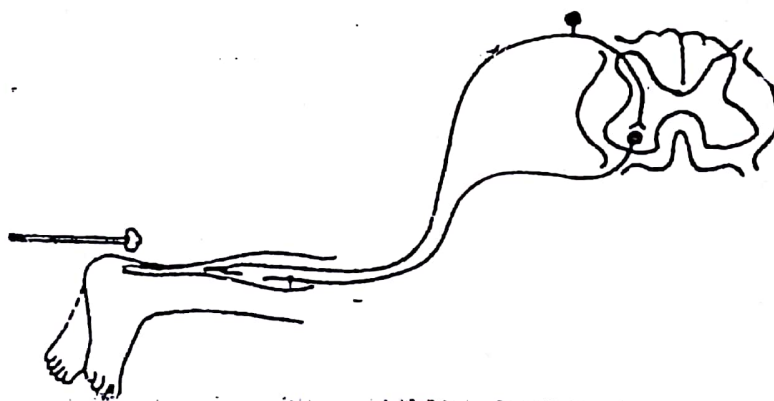
Two components of the stretch reflex

Moderate stretch $\longrightarrow$ Contraction

- Constant $\xrightarrow[\text{Muscle tone}]{\text{STATIC SR}}$ Constant
Bag Chain
Prim. & Second. Afferents
 More in antigravity muscles

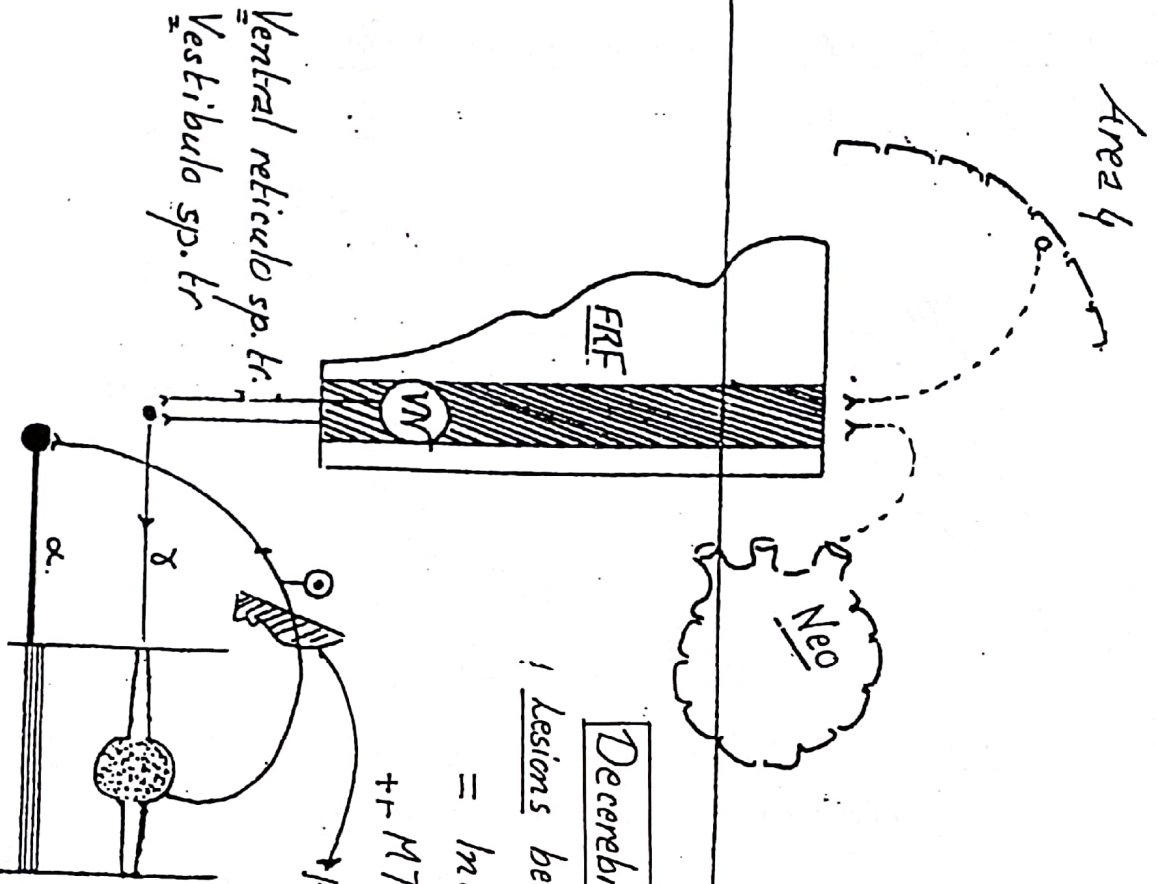


- Sudden $\xrightarrow[\text{Tendon jerk}]{\text{DYNAMIC SR}}$ Sudden
Bag
Prim. Afferent



Object is to keep constant LENGTH of muscle

Supraspinal Facilitatory Centres

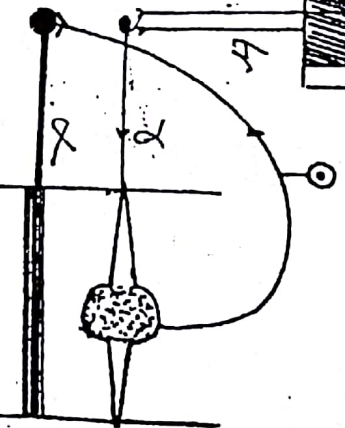


Decerebrate Rigidity

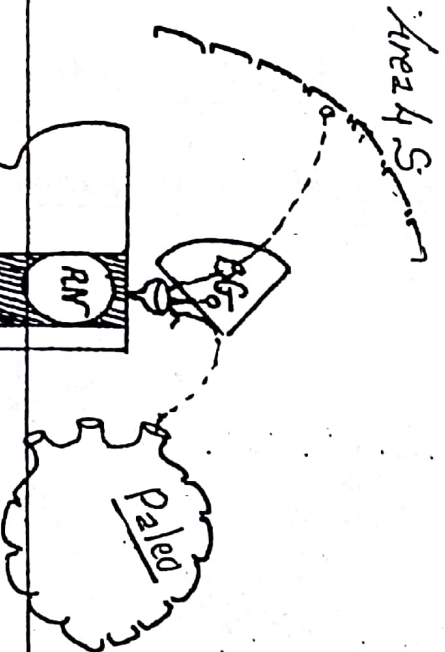
! lesions between pons & midbrain
= Internal capsule (man)
+/- MT of antigravity ms

Reflex nature

lateral reticulo sp. tr.
Rubro sp. tr.



Supraspinal Inhibitory Centres

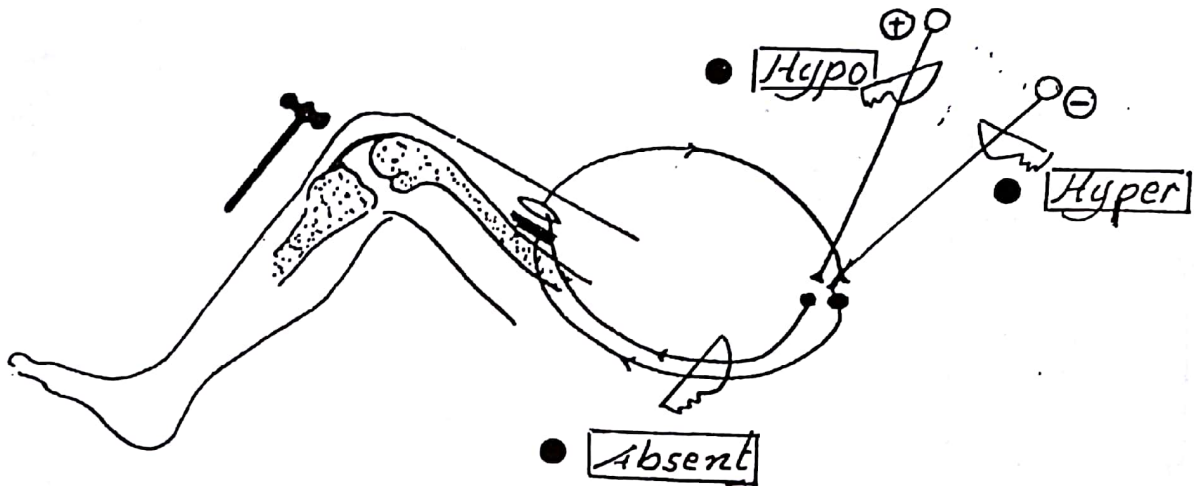


(no intrinsic activity)

Clinical importance of stretch reflex

Tendon jerk

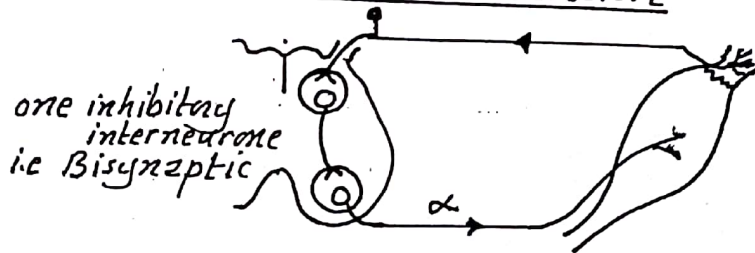
- Muscle tone



Inverse stretch reflex

Autogenic inhibition

Maintains constant tension

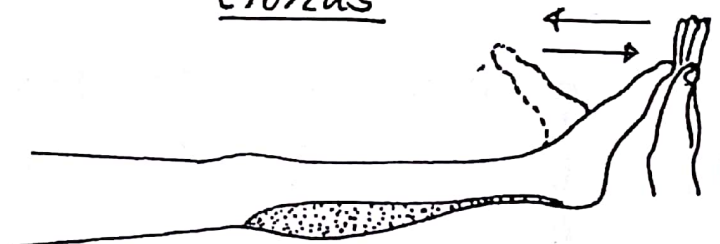
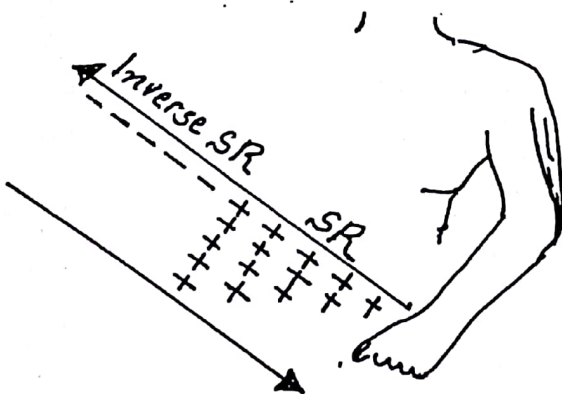


Findings with HYPERtonia due to UMN

Lengthening reaction

Clasp knife rigidity

Clonus



CNS

4

Complete TS of sp. cd

Above C5 <u>Resp. Failure</u>	Spinal shock	Recovery of reflexes
Below C5 <u>Diaphragmatic resp</u> <u>Quadriplegia</u> <u>Mid thoracic</u> <u>Normal respiration</u> <u>Paraplegia</u>	<u>Loss of all reflexes:</u> 1 <u>Stretch reflex</u> 2 <u>Flexor withdrawal R.</u> 3 <u>Defecation & erection</u> 4 <u>Micturition retention with overflow</u> 5 <u>ABP</u> -- 6 <u>VR</u> -- 7 <u>Skin</u> (bed sores/ulcers) 8 <u>Muscles</u> wasting	1 <u>Stretch reflex</u> : 1st to recover <u>Paraplegia in flexion</u> 2 <u>Flexor withdrawal R</u> : Babinski sign , foot, knee then hip. 3 <u>Deep reflexes</u> knee then ankle R 4 <u>Mass reflex</u> due to excitability - Flexion of LL - ABP ++ - Sweating - Lucidation unimpaired 5 <u>Autonomic reflexes</u> - ABP normal - automatic bladder & rectum - sexual (coitus) reflex 6 <u>Six months later</u> : - Paraplegia in extension - +ve supporting reaction - Mass reflex disappears
<u>Permanent loss below the level of</u> 1 <u>All sensations</u> 2 <u>All voluntary movements</u> <u>Reflexes pass in 2 stages:</u> - Stage of <u>spinal shock</u> - Stage of <u>recovery of reflexes</u>	<u>Cause & duration</u> : page 55 <u>Care of patient</u> : 1 <u>Catheterization</u> 2 <u>Rectal enema</u> 3 <u>Mobilization</u> 4 <u>Antibiotics</u> 5 <u>Nutrition & fluid balance</u>	<u>Cause of recovery</u> - <u>Demyelination hypersensitivity</u> - ++ <u>Collaterals</u> . CNS 5



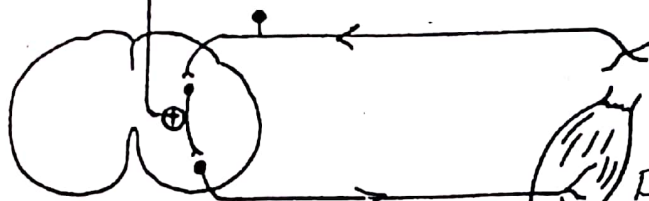
Stage of spinal shock



Cause

Sudden withdrawal of
supraspinal facilitation

→ Hyperpolarization
i.e. $++$ RMP 2 to 6 mV

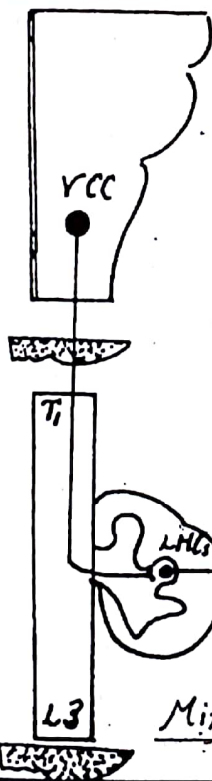


Depends on
ENCEPHALIZATION

Duration
Duration

Frog: minutes

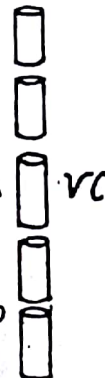
Man: 2-6 W



Drop in ABP

The higher the lesion
the lower the ABP

Maximal drop in ABP



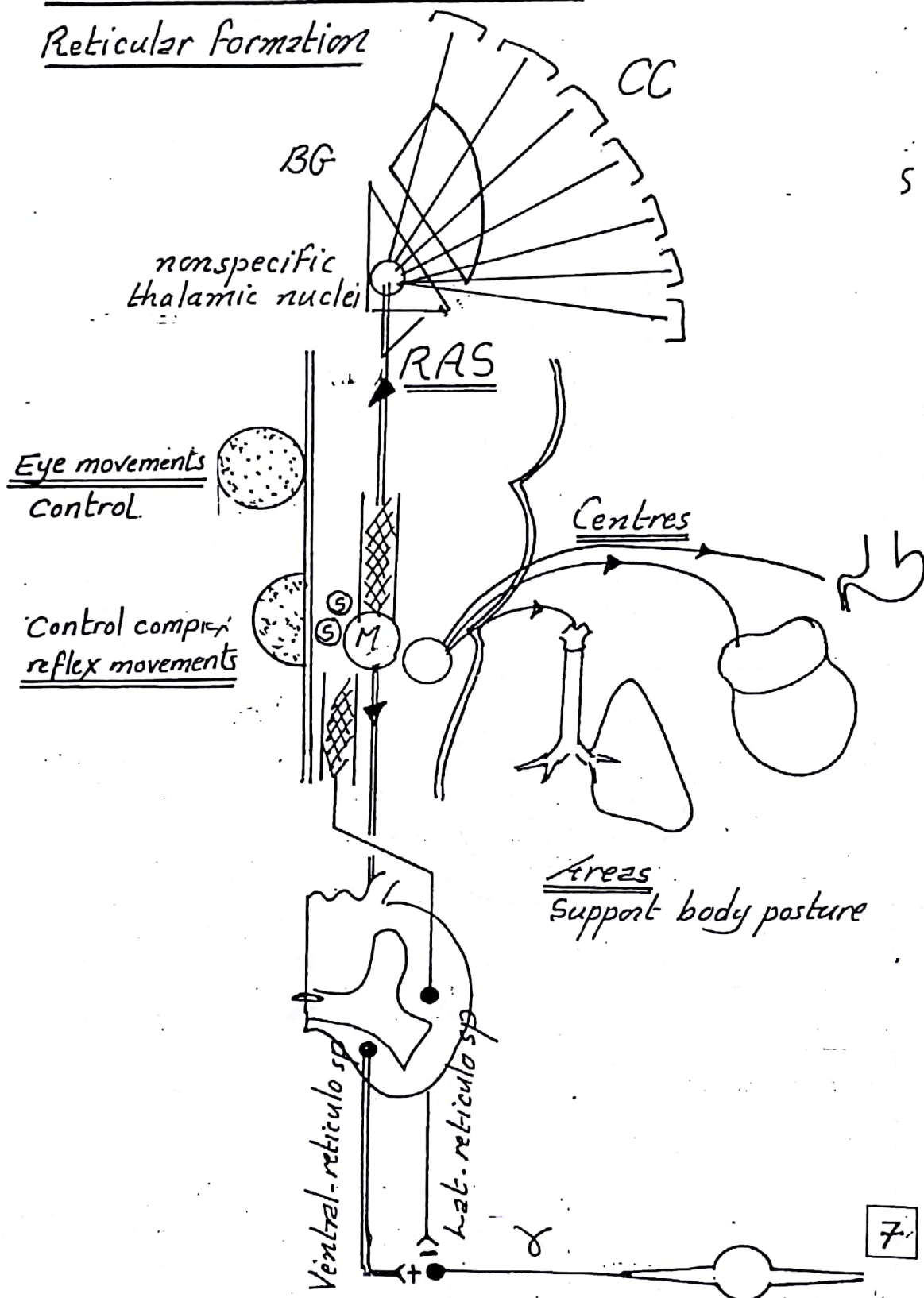
vc

Minimal drop in ABP

6

Motor Functions of brain stem

Reticular Formation



Labyrinth

Non auditory

Auditory

Cochlea

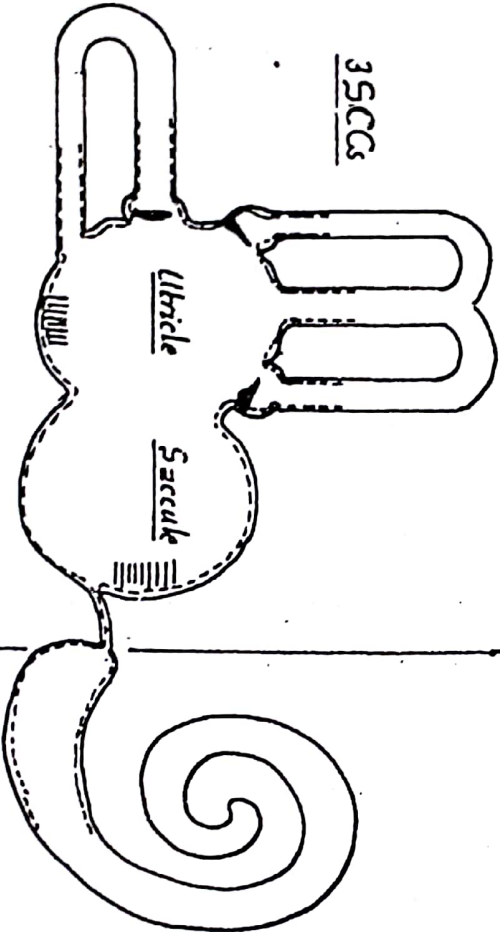
3 SCCs

Utricle

Saccul

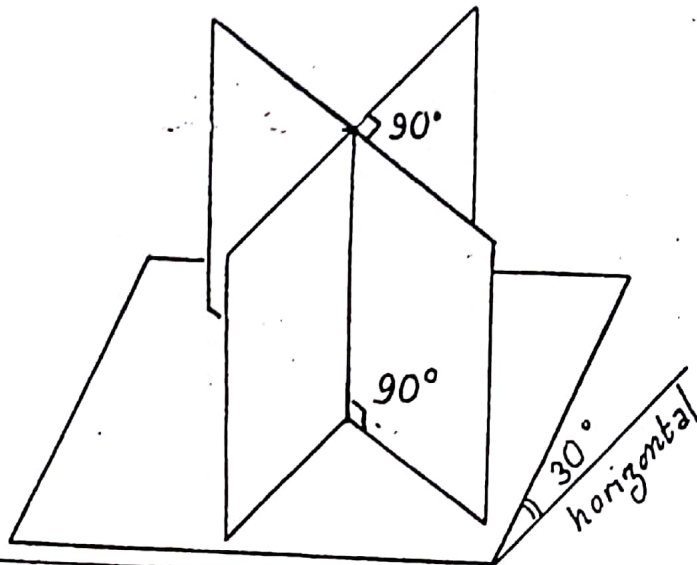
Equilibrium

Hearing



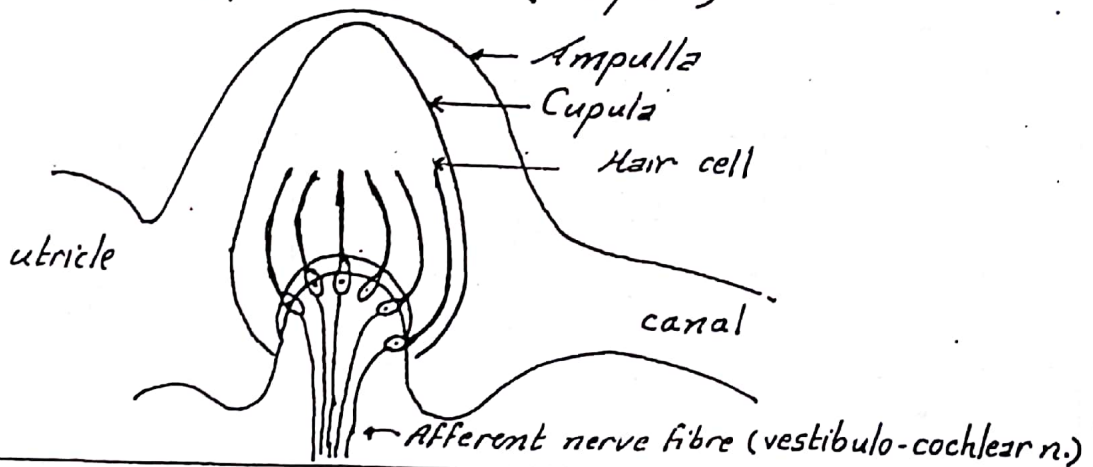
Semicircular canals

SCC₃



Crista ampullaris

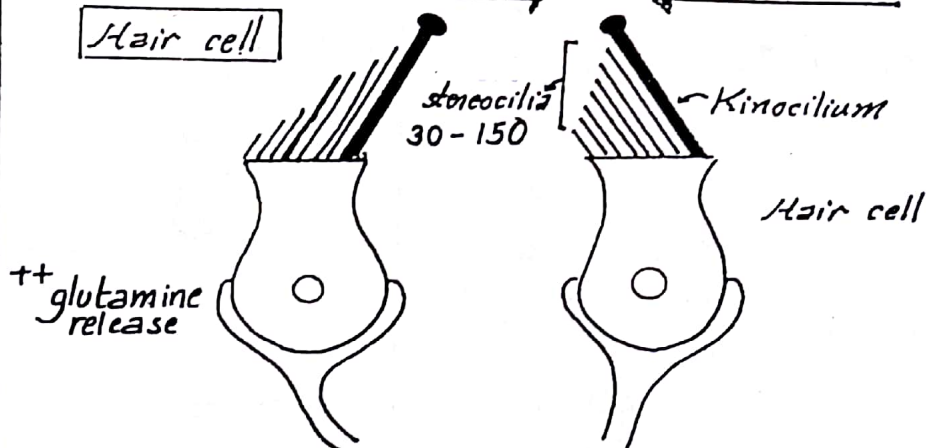
(receptors)



++ Discharge

-- Discharge

Hair cell



Angular acceleration 1 degree / sec.

● Before

Lt

Rt



(+)

utricle canal

RMP (+) -60 mV

● At the beginning

Inertia



(-)

Hypopol

Discharge

● Continuation with constant speed

Inertia overcome



(+)

endolymph

++ open time of K^+ & Ca^{++} ch.

Depol. -50 mV

++ Discharge 200/s



(+)

(+)

● At the end (stoppage)

Momentum

Vertigo



(+)

endolymph



(-)

● After few seconds



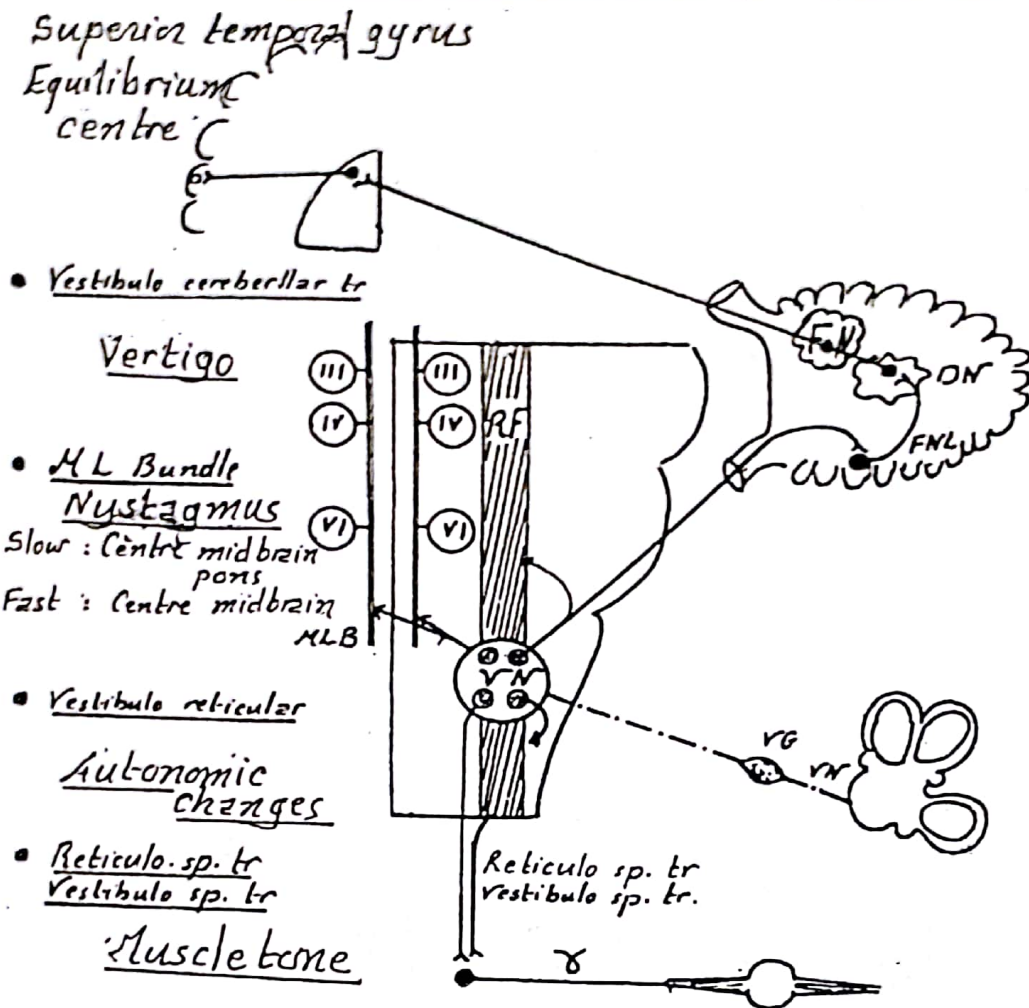
(+)



(+)

10

Nervous connection of non auditory labyrinth



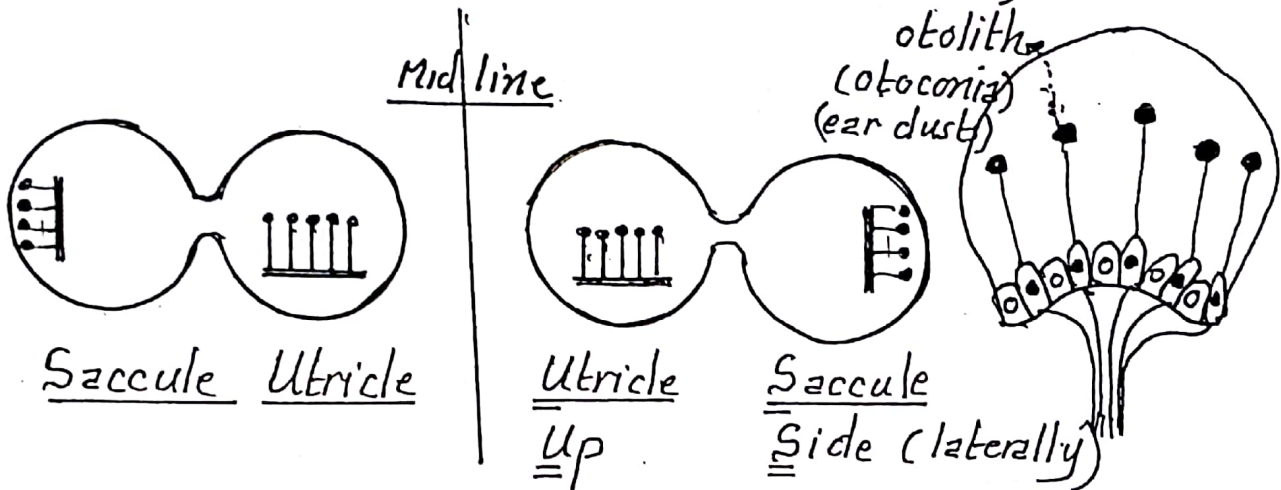
Methods of stimulation of SCCs

<u>Rotatory</u> rotation Horizontal 2 canals in 2 ears	<u>Caloric</u> convection current Vertical one canal in one ear	<u>Electrical</u> galvanic current 3 canals in one ear CNS
--	---	---

Utricle and Saccule

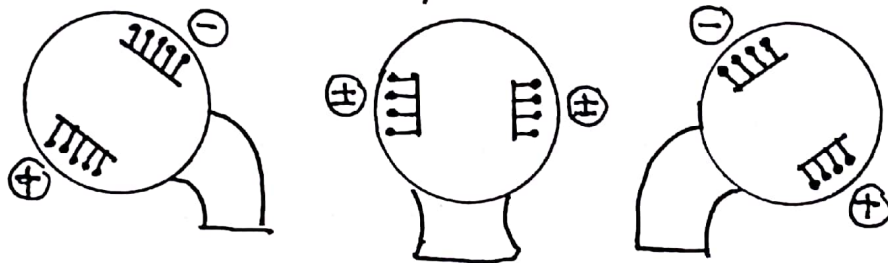
Receptor

Otolith organ (macula)



Functions of utricle & saccule

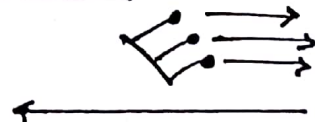
1. Maintenance of static equilibrium



2. Detection of LINEAR acceleration

Horizontal
Vertical

Utricle
Saccule



Postural reflexes

Maintain centre of gravity within area of support
Achieved by changing muscle tone i.e. stretch reflex

Functions 1. Maintain upright position

2. Postural background m. tone for voluntary movement.

Postural Reflexes

Static & Phasic

SP

Stretch

Crossed Extensor

+ve supporting

MEDULLARY statotonic

1 Tonic Labyrinthine

Otolith



2 Tonic Neck

Side



Up



Down

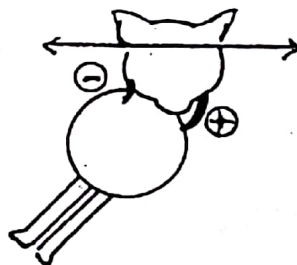
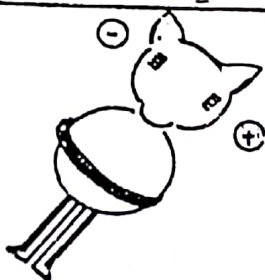


Neck propr.

1 Labyrinthine

RR

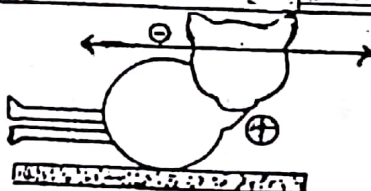
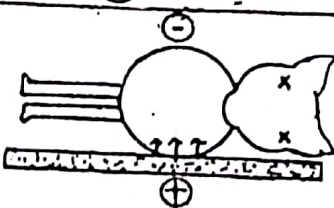
HEAD



2 Body

RR

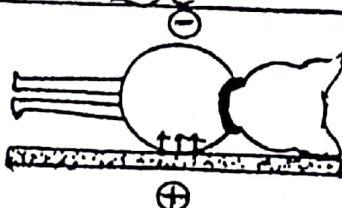
HEAD



4 Body

RR

BODY



3 Neck

RR

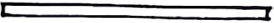
BODY

Stimulus: Unequal impulses from both sides

righting

MID BRAIN

13

<u>Cortical</u>	<u>Optical righting</u>	<u>Placing reaction</u>	<u>Hopping reaction</u>
			
		Rod, cones & exterocept	 M. spindles

Motor areas of CC

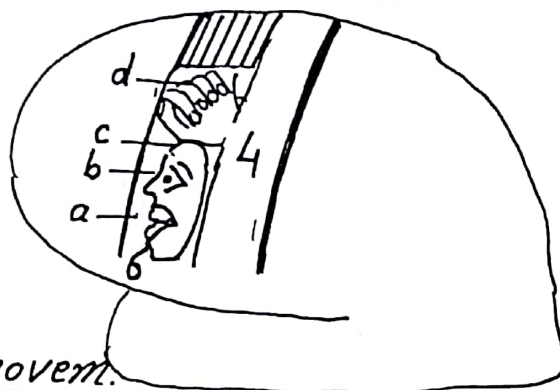
- 1 Prim. motor area Area 4
- 2 Supplementary motor area
- 3 Somatic sensory areas I & II
- 4 Premotor area

a Broca's

b Vol eye movem.

c Head rotation

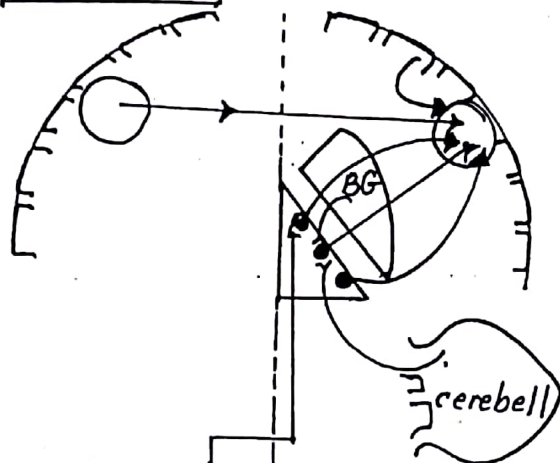
d Hand skill Lesion → Motor Apraxia



Connection of motor cortex

Afferents

2 x 2



Subcortical

Thalamic

Efferents Δ trs

1 Corticospinal tr

2 Corticobulbar tr

5, 7, 9, 10, 11 & 12

3 Corticonuclear tr

3rd 4th 6th

Extrapyramidal trs

LMN

85%

15%

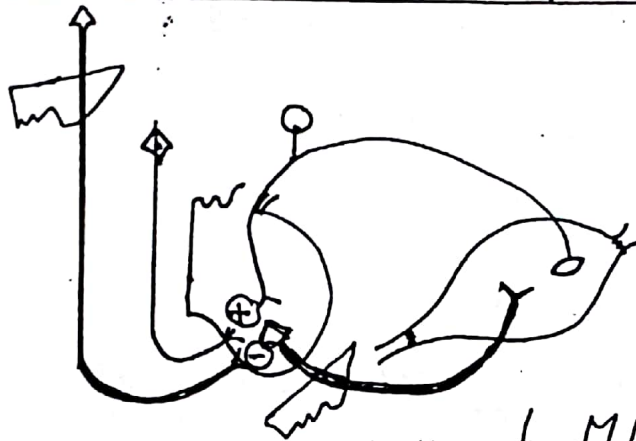
U M N

Exaggerated

MT & TJ

Absent

UMNL



LMNL

UMNL

LMNL

Post. limb of internal caps.
Usually opposite side
Usually contralat. hemiplegia
Recovery poor

Cause & Site
Side
Extension
Recovery

Neurone Nerve N-M junction.
Always same side
Usually localised
Recovery may occur

Hyper tonia
Spastic paralysis
Hyper reflexia
Present

Muscle tone
Paralysis
Deep reflexes
Clonus & Clasp knife R.

Atonia
Flaccid paralysis
Absent
Absent

Absent
+ve Babinski sign

Superficial reflexes
Plantar reflex

Absent

Mild or no
Absent

Muscle wasting
M. Fasciculation

Marked
Present

Normal electrical resp:

Reaction of degeneration

Present

Lesion of post. limb of internal capsule:

- 1 | | emiplegia.
- 2 | | emianesthesia.
- 3 | | emianopia.
- 4 | | hearing is slightly affected.

Basal ganglia

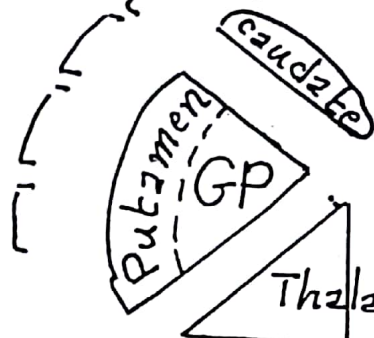
BG

SLOW motor program

Formed of 3 major nuclei

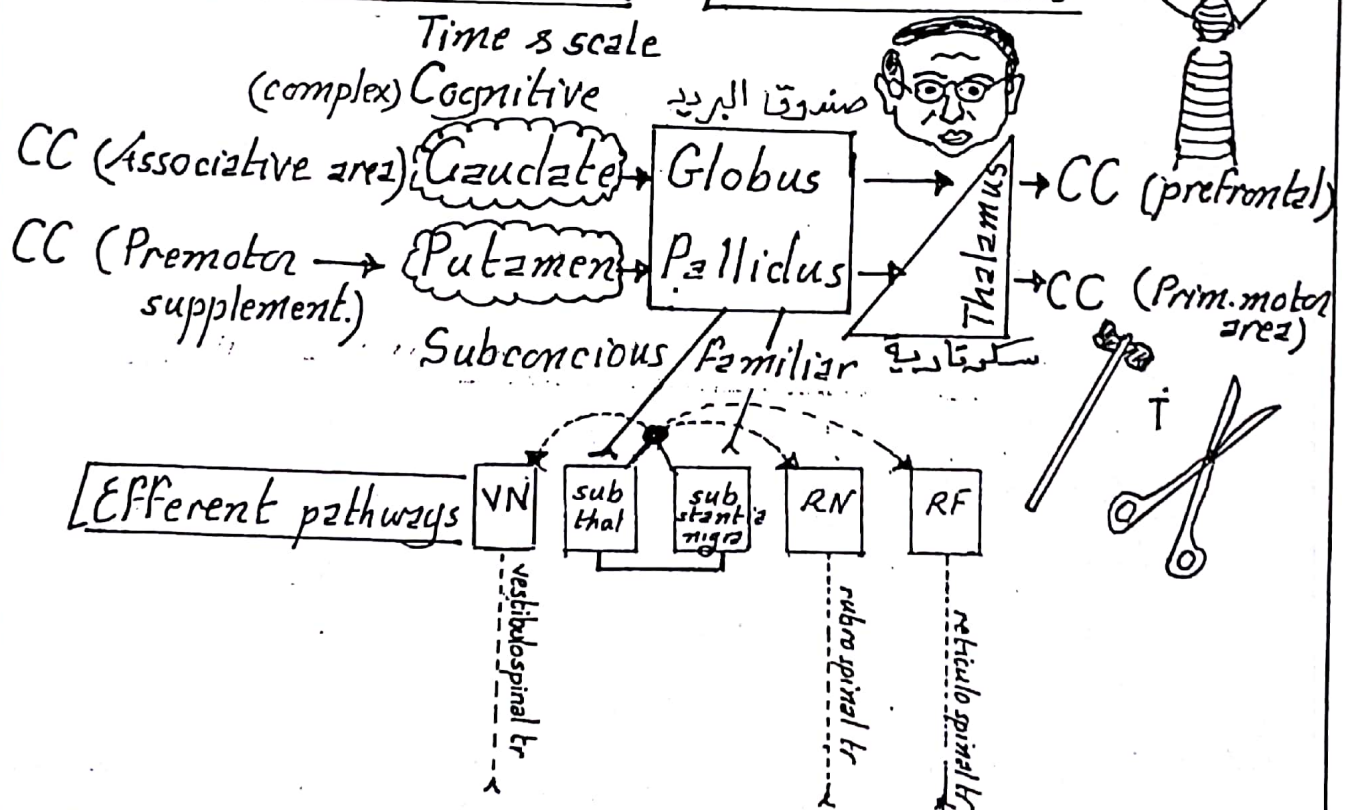
2 related nuclei

subthalamus
substantia nigra



Neostriatum Lenticular nucleus
Caudate Putamen Globus pallidus

Nervous connection of BG : 2 main circuits



Ch. tr CC • A. choline → ⊕ Neostriatum
• GABA → ⊖ Substantia nigra
• Dopamine → ⊖

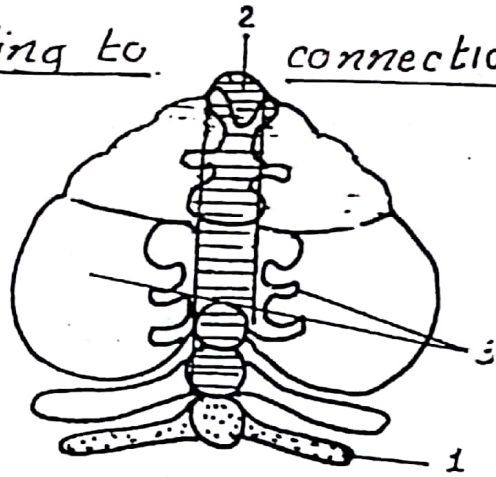
Chorea A. choline
GABA
result inhibition
Parkinsonism Dopamine
result excitation

Cerebellum

Functional anatomy : According to connection

- 1 Vestibulo (Arch) cerebellum
= Flocculo nodular lobe
Equilibrium
- 2 Spino-(Paleo) cerebellum
= midline area (vermis)
Coordination
- 3 Cerebro (Neo) cerebellum
= lateral zones of hemisphere
Planning & programming
Facilitatory to m. tone

AHGsaCC Vestibule



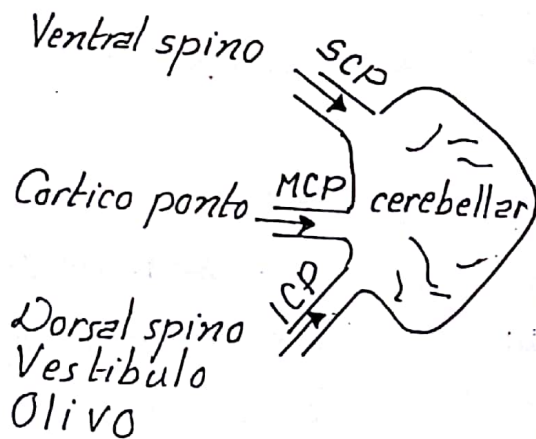
Cerebellar connections

- Afferents (Climbing & Mossy) end in cerebellar cortex (3 layers) and synapse with Purkinje cells.
- Axons of Purkinje cells carry impulses to cerebellar nuclei (3 Dentate, Interpositus and Fastigial).

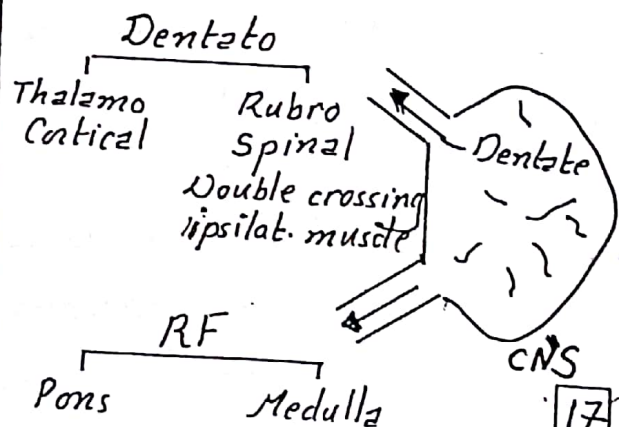
Topographic representation

- Ant. lobe inverted
- Post. lobe erect
- Lat. zone no

Afferents



Efferents



Functions of cerebellum

Silent area

Coordinate Rapid motor activity.

A In voluntary movements

1 Servo comparator Function

Closed Feed back circuit between

motor areas of CC & intermediate zone of cerebellum.
Cortico ponto DENTATE thalamo cortical
Cerebellum

1 Informed

a CC intention viz

Cortico Ponto Fastigial tr

b Muscles performance viz

Spino cerebellar tr

2 Compares

3 Corrective signals to CC Fastigial thalamo Cortical tr

2 Braking effect Stops movement at precise intended point

Mechanism Assesses rate & Calculates length of time

Inhibits agonists & Excites antagonists

3 Planning & timing Function

Lateral zone plans for NEXT while present mov. is occurring

B Other Functions

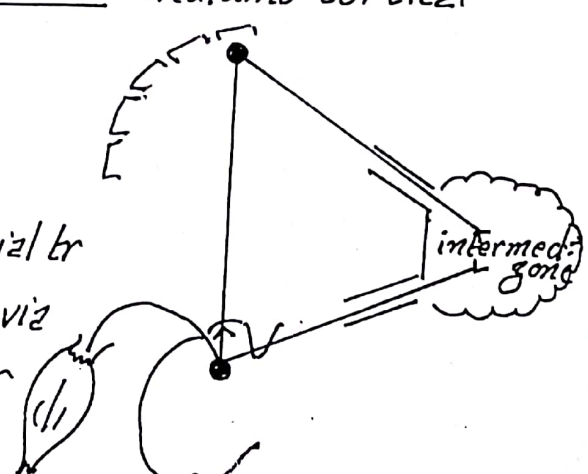
1 Equilibrium : Flocculo nodular lobe

Vestibular apparatus → " → brain stem

→ viz reticulo & vestibulo sp. trs changes axial & girdle
muscle tone maintains equilibrium

2 Muscle tone Neocerebellum Facilitatory

Paleocerebellum Inhibitory



Clinical abnormalities of cerebellum

<u>Lesion</u>	<u>In deep nuclei</u> or <u>Cortex</u>	permanent effect temporal effect
<u>Effects</u>	<u>SAME SIDE</u>	

ATAXIA Incoordination of voluntary movements
In absence of motor lesion
ie no UMN/L no LMN/L

Other manifestations

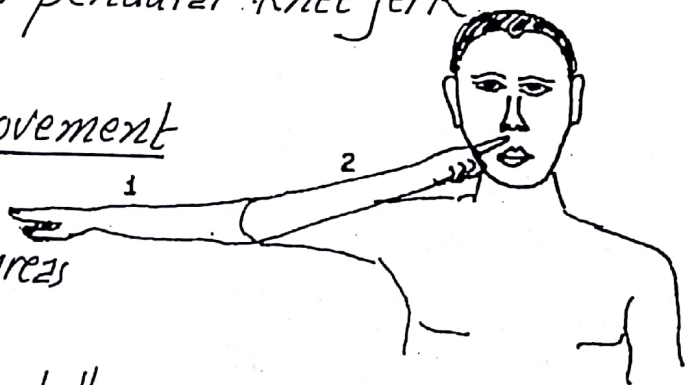
- 1 Dysmetria
- 2 Dys diadokokinesia
- 3 Dys arthria
- 4 Decomposition of movements
- 5 Drunken gait & Disturbance of posture
- 6 Kinetic tremors
- 7 Eye ball tremors nystagmus
- 8 Rebound
- 9 Hypotonia & pendular knee jerk

N.B Control of voluntary movement

- 1 Comands
Cortical association areas

- 2 Plans
CC (premotor) BG Cerebellum

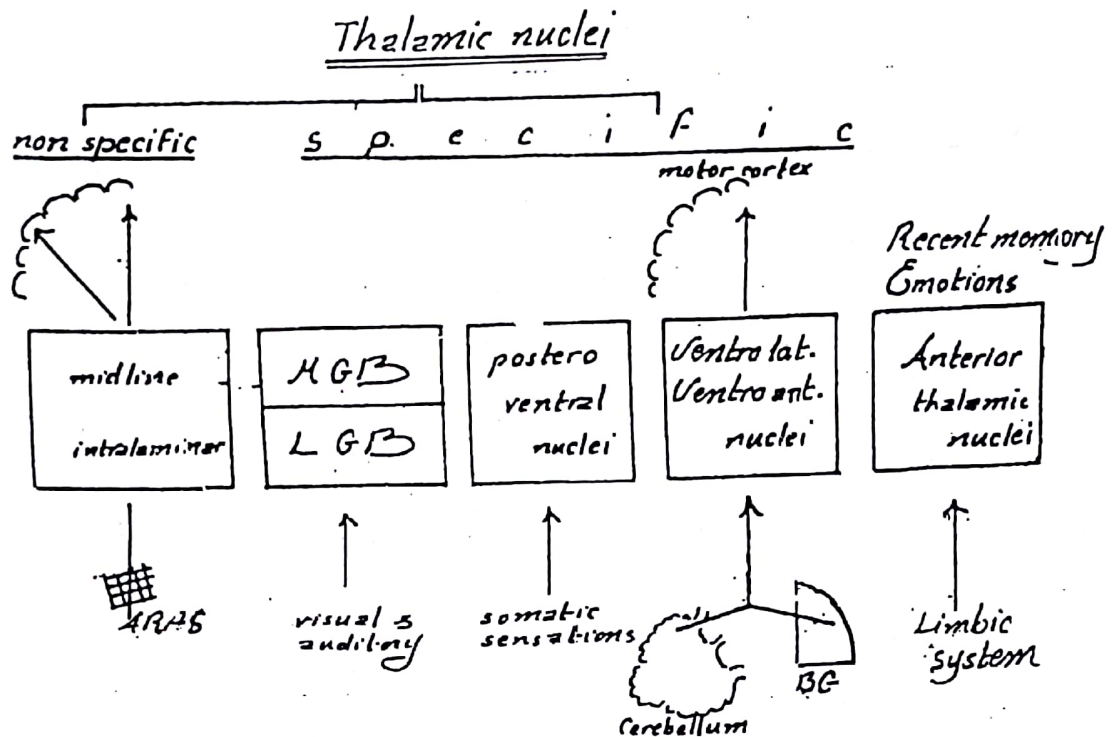
- 3 Excution
 Δ tr & extra Δ tr



Finger to nose test

Thalamus

Highest subcortical SENSORY centre



Functions of the thalamus

- 1 Relays station for all SENSATIONS except olfaction.
- 2 Non specific thalamic nuclei → excitability of CC
- 3 Memories coding, storing & recalling
- 4 Relays autonomic & emotional signals to hypoth. & limbic system
- 5 Relays motor signal from BG & cerebellum to motor cortex

Thalamic syndrome

- Cause thrombosis of arterial supply
- Damage posterior (not medial or anterior) nuclei
- Results
 - 1 Hemianæsthesia on opposite side
 - 2 2nd Hyperalgesia pain threshold is increased. but when reached pain is severe and prolonged.
 - 3 Sensory ataxia
 - 4 Emotional disturbance.

Electrical activity of the brain

① Evoked potentials

- Def Potential changes in CC caused by receptor stimulation
- Recording Person is anaesthetised
Exploding electrode over the excited cortical area.
- Primary EP Over excited area
5-12ms
+ve -ve
- Diffuse secondary EP Over most of the cortex
20-80ms
prolonged +ve
from nonspecific thalamic nuclei
- Importance to map the brain.
SSEP, AEP, VEP: diagnosis of certain diseases especially VEP
e.g multiple sclerosis

② Electroencephalogram EEG (spontaneous potential)

- Defin: Record of spontaneous electric activity of the brain
- Recording Applying electrodes on the scalp of patient
Calm room
Comfortable temp
Complete physical and mental rest
- Waves Intensity 0 - 200 mV
Frequency 1 - 50 Hz

Look table page

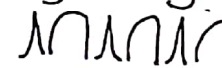
N.B If Theta & delta waves occur in an adult during waking state = abnormality or hyperventilation

Desynchronization of alpha block.: When eyes are opened.
 α wave is replaced by faster low volt. irregular β waves.

Types of normal EEG waves 4 types of normal EEG waves

	Alpha	Beta	Theta	Delta
1. Frequency	8 - 13 Hz	18 - 30 Hz	4 - 7 Hz	1 - 3 Hz
2 Amplitude	50	20	100	100
3 Age	Adult	Adult	Child	Infancy
4 Recorded at	Parieto-Occipital	Frontal	Parietal/temp	
5 State of the person	Physical & Mental rest Awake Closed eyes	Intense activation of CNS i.e. during thinking & tension	Or in adult during emotional stress	Or in adult during sleep

• Clinical uses of EEG :

- ① Localization of brain tumors Large tumour -- electric activity at the region. Other tumours excite → very high voltage.
- ② Diagnosis of epilepsy
 - a Grand mal „ : High voltage spike followed by slow waves
 - b Petit mal „ :  3 / second.
- ③ Diagnosis of sleep disorders.
- ④ Confirmation of brain death.

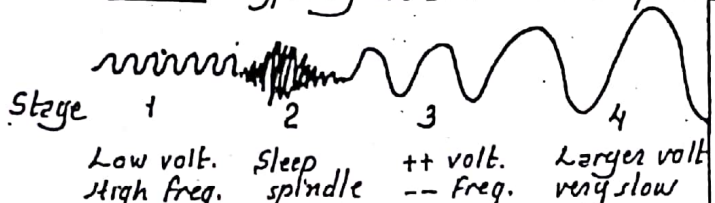
Sleep

- Defn loss of consciousness from which a person is aroused by proper stim.
- Types of sleep 2 Look table page
- Distribution of sleep stages

A young adult, enters non REM stage 1 & 2 spends 70-100 min. in stage 3 & 4 sleep lightens → REM period. (cycle 90 min)

4 to 6 REM periods per night

% of REM sleep -- in old age.

Non rapid eye mov. (non REM) Slow wave sleep	Rapid eye movement (REM) paradoxical sleep
It occurs at the start of sleep 80% Duration/cycle 90 min	After 4th stage of non REM 20% 20 min
<u>EEG</u> Typically theta & delta with spindles 	Beta waves  irregular, low volt., high freq. = alert state (beta waves)
<u>Eyes</u> Deviate up	Rapid eye movement
<u>Talking & walking</u> present	absent
<u>Dreams</u> Present not remembered	Present & remembered.
<u>Threshold of awaking</u> Low	High & difficult to awaken.
<u>HR, resp. rate, ABP & RR</u> Decreased	Increased.
<u>Muscle tone</u> Hypotonic	Marked hypotonia

• Mechanism of sleep

- (A) Slow wave sleep is produced by 3 subcortical regions (zones)
- 1 Diencephalic zone in post. hypothalamus
 - 2 Medullary synchronizing zone in RF of medulla
 - 3 Basal forebrain sleep zone.
- Ch. transmitters -- conc. of serotonin, ++ conc. of adenosine & release of PG D₂ in preoptic area of hypoth. → slow wave sleep
- (B) REM sleep is triggered by mechanism in pontine RF.
Cholinergic discharge by these neurones → REM sleep.

• Sleep Disorders

- 5 Insomnia Adult Due to psychological factors e.g. anxiety
- 4 Somnambulism Sleep walking Child eye opened avoid obstacles
- 3 Narcolepsy Irritable sleep starts with REM sleep.
- 4 Sleep apnea Obstruction of airway awakens the person
- 5 REM behavior disorder Act out their dream No hypotonia Rare (23)

Hypothalamus

maintains homeostasis

Afferents From :

Prefrontal area, limbic lobe, amygdala
hippocampus, nonspecific thalamic nuclei
globus pallidus & ascending sensory trs

Efferents to :

Neurohypophysis,
ant thalamic nuclei &
brain stem RF

Functions of hypothalamus

① Autonomic function :

- Lateral & dorsomedial nuclei → symp effects
- Superior anterior hypothalamus → parasymp

② Food intake :

Appetite
Lateral Feeding Ventromedial Satiety

③ Temperature regulation :

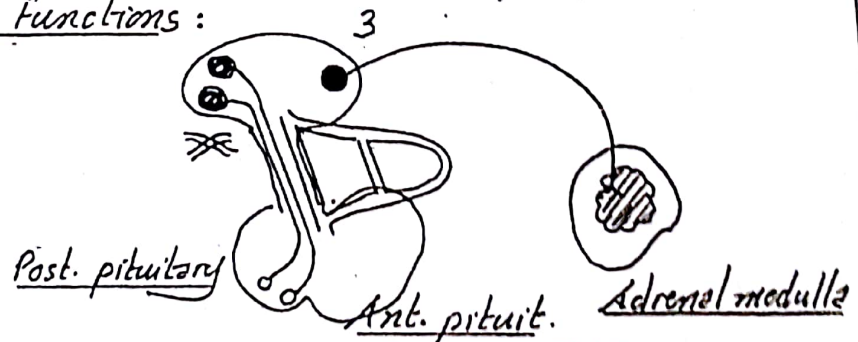
Anterior (heat loss)

Posterior (heat gain)

④ H₂O balance :

- H₂O gain : Thirst centre in lateral hypoth
- H₂O loss : ADH Supraoptic nuclei

⑤ Endocrinal functions :



As a part of limbic system :

6 Behavioral function pleasant or unpleasant response

7 Defensive reaction rage & fear expression

Relation to cyclic phenomenon :

8 Circadian rhythm Light dark cycle

9 Wakefulness & sleep As part of RAS Stim of ant. hypoth → sleep

10 Control of sexual function via influence on ant. pituitary

Limbic system

behavior associated with emotions

• Anatomical consideration

Limbic system consist of

[1] Allo-cortex ring on medial side around corpus callosum

[2] Subcortical nuclei : e.g hypoth, thalamus, BG, amygdala

Communications exist between its different components & with neocortex.

Prolonged after discharge in its circuits → prolonged emotions.

• Functions of limbic system

[1] Olfaction

[2] Sexual behaviour reflex regulated by limbic system & hypoth.
Unlike animals (instinctual or hormonal) In humans it is complex.

[3] Autonomic behavior that accompany emotional state.

[4] Control of emotions Fear & rage are related phenomena.

Fear avoidance reaction Centre Hypoth. & amygdala

Rage fighting reaction. Centre Hypoth. & amygdala
inhibited by neocortex or ventromedial nuclei of hypoth.



(5) Motivation

Limbic system has 2 areas:

[a] Areas give rewarding sens.
between frontal cortex via
hypoth to midbrain

[b] Areas give punishment
lat. portion of post. hypoth

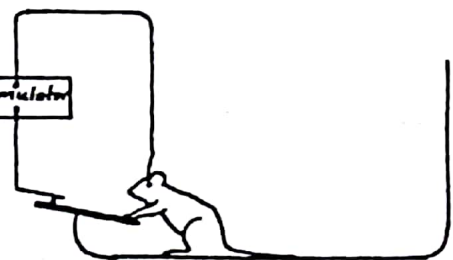
Reward & punishment are important for

[a] Motivating our behavior

to gain reward or avoid punishment.

[b] Learning

reward or punishment → reinforcement



Higher brain function

Learning & memory are related → alter behavior

Memory is divided into: ^{repeated}

Non declarative = reflexive = Implicit

- Unconscious recall
- Doesn't involve hippocampus
- It includes:
 - 1 Skills & habits
 - 2 Priming e.g. Physio.....
- Divided into

Declarative = recognition = Explicit

- Conscious recall.
- Depends on hippocampus & medial temporal lobe
- Types:
 - 1 Episodic For events
 - 2 Semantic For words, rules

a) Non associated learning

- 1 Habituation repeated S → -- response
 - 2 Sensitisation repeated S → ++ response
- if coupled with unpleasant or pleasant stimulus

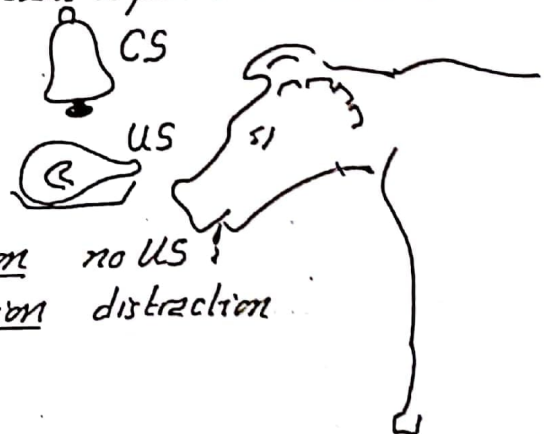
b) Associative learning

1) Classic conditioning

Pavlov experiment

Characteristic:

- internal inhibition no US
- external inhibition distraction
- reinforcement



2) Operant conditioning

≠ classic Here the animal is active (perform a task)

Explicit memory & some implicit memory involve:

<u>Immediate (sensory) memory</u>	<u>Short term memory</u>	<u>Long term (fixed) memory</u>
- Few seconds or minutes - initial stage for memory - Fades or changes to other types - <u>Molecular base</u>: ++ synaptic transmiss. • <u>Reverberating circuits</u> • <u>Post tetanic facilit.</u>	- Few minutes or hours - <u>Characteristics</u> 1 rapid recall 2 easily displaced 3 working memory - <u>Molecular bases</u>: - Facilitated pathway (temporary memory trace)	- hours to years or life long - <u>Characteristics</u> 1 not instantaneous 2 resist disruption 3 develops from other forms - <u>Molecular base</u>: - Structural change in presyn. terminals (memory engram)

Consolidation of memory trace = encoding the memory
 & makes it resistance to erase.

It involves the hippocampus & its connection.

Centres for memory:

1. Implicit memory stratum of BG & cerebellar folliculus
2. Explicit memory
 - short term in hippocampus
 - long term in neocortex & amygdala (emotional aspects)

Clinical links

1. Amnesia
 - a. retrograde: before injury.
 - b. antograde: after injury.
2. Alzheimer's disease progressive loss of short term memory
 followed by loss of cognitive and other brain functions
 Cause Degeneration of hippocampal neurones

Speech and language

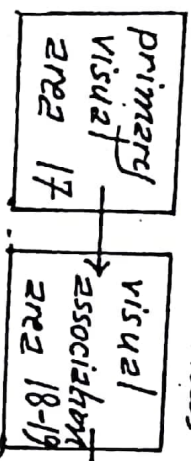
Cerebral hemispheres

Categorical	Representational
Left	Right
<u>Site</u> 	
<u>Functions</u> 1. Analytical process 2. Language function	1. Recognition of faces 2. Identification of objects 3. Understanding & interpret music
<u>Lesion</u> <u>Aphasia</u> (language & speech disorders)	<u>Agnosia</u> Inability to understand the color of speech or to tell a joke or story

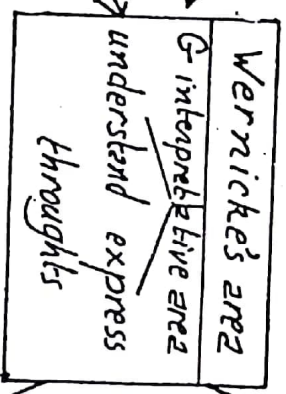
Mechanism of speech

Highest function

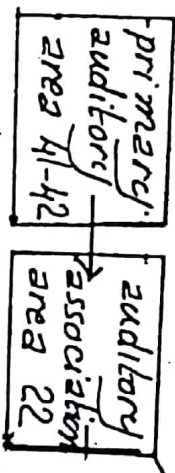
Visual aphasia Ability to understand spoken words or word produce written words
blindness



Fluent aphasia



Auditory aphasia or word deafness

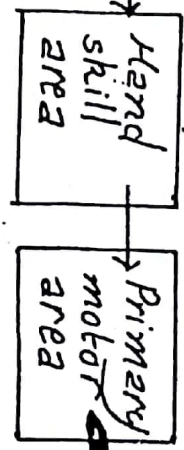


Global aphasia

extensive categorical hemisphere Amusia representational hemisphere.

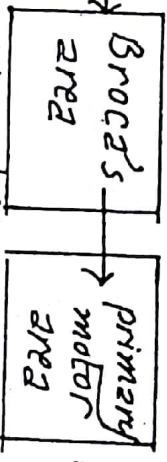
Aphasias

no visual auditory motor Defect



Motor aphasia Agraphia

Non Fluent aphasia



Motor aphasia OK, Yes or No



Complementary Specialization of the hemispheres :

	Categorical	Representative
<u>Site</u>	Left hemisphere. 96% of Rt handed 70% of Lt handed	Right hemisphere.
<u>Function</u>		
1 <u>Language</u>	grammar vocabulary literal	intonation ^{تنجويد} prosody ^{قوافي} pragmatic context ^{سياق} jokes, stories, colour of speech
2 <u>Calculation</u>	exact	approximate
3 <u>Memory</u>	Recall	Recognition of faces Identification of objects
4	Analytical	Holistic Understands interpret music
<u>Lesion</u>	<u>Aphasia</u> i.e Abnormality of language In absence of deafness, blindness or paralysis	1 <u>Asteriognosis</u> 2 <u>Agnosia</u> inability to recognise object by particular sensory modality e.g. <u>Amusia</u> 3 <u>Unilat. inattention & neglect</u>

Pierre Paul Brocz (1861): Pt named Tzn had syphilis in Brocz's area

Karl Wernicke (German): Area in Lt superior temporal gyrus.

Left handed individuals : - higher % of artists, musicians & mathematicians
7% - Slightly but significantly shorter life span

- Dyslexia (impaired inability to learn) 12 times that of Rt hand

	Human brain	Chimpanzee brain	Monkey brain
Size	16	4	1

Types of aphasia

1 Nonfluent : Lesion in Broca's area

Mild lesion Speech is slow & words are hard to come.

Severe lesion Speech is limited to 2 or 3 words

2 Fluent : Lesion in Wernicke's area

Pt speaks normally or even talk excessively. Speech is full of jargon and neologisms that make little sense

Again Pt fails to comprehend the meaning of spoken or written words

3 Conduction : Lesion in arcuate fasciculus connecting Wernicke's & Broca's areas

Pt can't put parts of words together or conjure up words

4 Anomic . Lesion in angular gyrus

Pt has no difficulty with speech or auditory information.

Pt has trouble in understanding written language or picture

5 Dyslexia 5% of population have impaired ability to read

It is inherited disorder. Cause is unknown

However, there is -- blood flow to angular gyrus

6 Selective defects e.g lesion in Lt temporal pole (area 38) leads to

Inability to retrieve name of places or persons, but preserve ability to retrieve common names, verbs & adjectives

7 Global i.e more than one form of aphasia is often present.

Speech is scant as well as nonfluent.

Note Writing is abnormal in all aphasias & in case of deaf persons, they lose ability to communicate in sign language

Lesion in representational hemisphere leads to impaired ability to tell a story or make a joke. Again, Pt ability to get the point of joke and to comprehend the color of speech is impaired.

Lesion in right inferior temporal lobe → prosopagnosia i.e inability to recognise face. Pt recognises people by their voices 30